The Journal of HEREDITY

SILKY DOVES

(SEE PAGE 37)

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Drosophila Pupal Lethals

Tongue Movements in Twins

DDT-Resistant Inbred Houseflies

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The

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CONTENTS

Spermatogenesis in an Elaterid Beetle
By Stanley G. Smith 3

Genetics of DDT-Resistance of an Inbred Line of Houseflies
By Elizabeth Thomas Lichtwardt 11

A Study of the Inheritance of Certain Tongue Characters in 72 Pairs of Negro Twins
By J. Warren Lee 17

Pupal Lethals in *Drosophila melanogaster*
By Frank Seto 21

Whiteside, a New Mutation in *Peromyscus*
By William B. McIntosh 29

Inheritance of Resistance to Pink Root in the Onion
By Henry A. Jones and Bruce A. Perry 33

Silky Plumage in the Ring Neck Dove
By Wilmer J. Miller 37

Intra-Species Relationships and Lysenko's Concept of a Species
By Constantine Sorokin and W. Gordon Whaley 41

Notes and Reviews

Translocations in Naranjilla, an Ecuadorian Solanaceous Fruit
By Patricia Sarvella 19

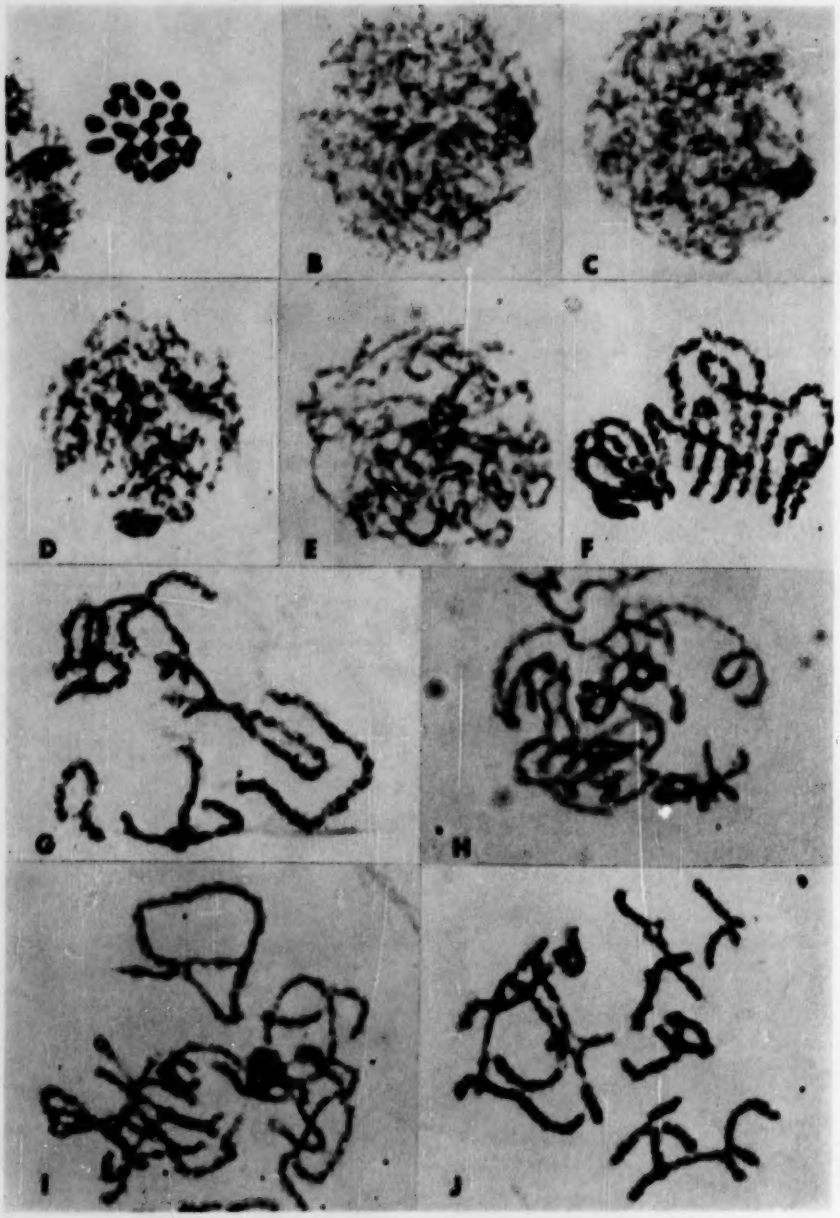
A New Publication Policy 27

Chemogenesis? 32

Actions of Radiations on Living Cells (Rev. of Lea: *Actions of Radiations on Living Cells*)
By W. Ralph Singleton 34

Breeding Beef Cattle for Unfavorable Environments (Rev. of Rhoad: *Breeding Cattle for Unfavorable Environments*)
By Bruce L. Warwick 35

Counseling in Medical Genetics (Rev. of Reed: *Counseling in Medical Genetics*)
By Eldon J. Gardner 36



SPERMATOGONIAL METAPHASE TO MID-DIPLOTENE

Figure 1

A—Metaphase from the fifth series of spermatogonial mitoses. *B* and *C*—Leptotenes showing precociously condensing X chromosome. *D*—Side view of the developing 'bouquet' at early zygotene. *E*—Polar view of initiation of zygotene pairing. *F*—Pachytene 'bouquet' comprising nine bivalents and the pear-shaped X. *G*—Disarranged pachytene showing nine bivalents (note relational chromosome coiling in rightmost) and the strap-like X (center). *H*—Very early diplotene. *I*—Metacentric ring bivalent at very early diplotene. *J*—Later diplotene showing spirals in the X (at 11 o'clock) and in regions of the autosomes.

SPERMATOGENESIS IN AN ELATERID BEETLE

STANLEY G. SMITH*

THE almost 500 species and subspecies of Coleoptera that have been studied since the pioneer investigations of Miss N. M. Stevens, nearly fifty years ago, reveal the existence in the order of an unusual degree of cytological diversity, perhaps at least equal to that found in any comparable group of animals. The norm may be described as a bisexual species possessing nine pairs of autosomes, both rod shaped (acrocentric) and V shaped (metacentric) and associated during meiosis by one or two chiasmata, and an XX (♀) : XY (♂) sex-determining mechanism in which the Y chromosome is minute and paired with the X in the form of a parachute (Xy_p).

Interspecific numerical deviations from this type range from five to 28 pairs of autosomes[4]. Intraspecific variation has been encountered in seven species in five different families, due, in five of the species, to their possession of supernumerary, genetically inert chromosomes[7,12] and, in two, to "centric fusion" (translocation between two non-homologous autosomes followed by loss of one derivative)[8,9]. For the Xy_p sex-chromosomes of the orthodox male, there may be substituted a single X, a neo-XY pair (i.e., a pair in which the X has "fused" with an autosome, the homologue of which has become the new Y chomosome), a multiple sex-chromosome mechanism (i.e., 2, 3, or more X chromosomes along with the Y)[1,2,6], or an indistinguishable pair presumably derived from a neo-XY type.

The single X in XO males goes undivided to one of the poles at the first meiotic division and divides equationally at the second (prereduction) in all XO species except those of the Lampyridae[5], in which the sequence is reversed (postreduction). Where there are two or more sex chromosomes, however, their synchronization in movement with the separating autosomes at the first anaphase finds an exception only in one tenebrionid, *Zopherus haldemani* Sallé. Therein the X and y approach opposite poles (anaphasic precession) while the autosomal bivalents are still congressed on the metaphase plate. This species is further, and perhaps correlatively, exceptional in that the autosomal bivalents undergo a pronounced premetaphase stretch before congressing on the equatorial plate.

Two further, phylogenetically different, types of reproduction are found in the order. Both involve parthenogenesis. In one, males are haploid and females diploid, as they are in bees and wasps; in the other, males are wanting and the females may be diploid, triploid, tetraploid, or even pentaploid[11].

In the majority of species—those with especially small chromosomes—the association of autosomes in homologous pairs at first metaphase is assumed to be by chiasmata, an inference based on observation of the larger chromosomes of the most favorable species. Assuming all conjugated chromosomes to be so joined, the number of chiasmata appears to be determined largely by the size of the conjoined elements, subject however to the presence or absence of heterochromatin. Moreover, if heterochromatin is present, its site along the chromosome (usually if not always procentric) interferes with the position in which chiasmata may be formed. Despite the foregoing, species are known in which heterochromatin, detectable by its heteropycnosis (differ-

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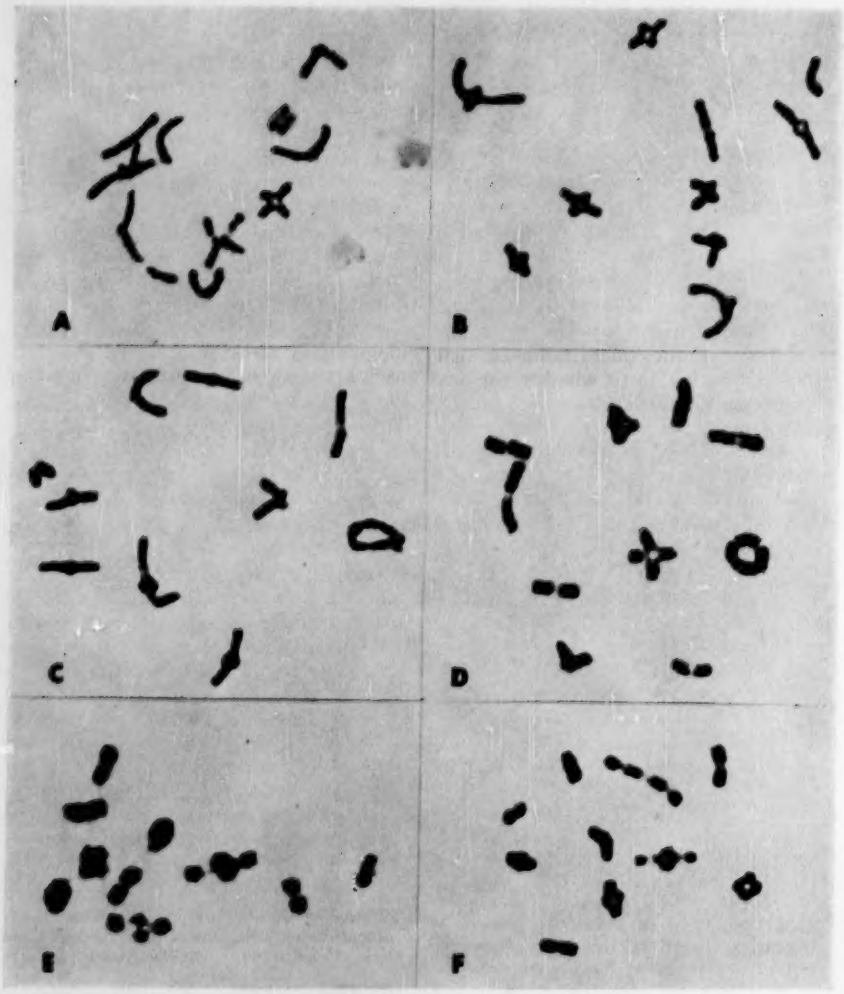


MID-DIPLOTENE TO LATE DIAKINESIS

Figure 2

A—Mid-diplotene showing the X with spirals and the metacentric bivalents with accentuated constrictions. *B* and *C*—Two later diplotenes from the same cyst, one with and the other without a metacentric ring bivalent. *D*—The only metacentric ring bivalent found in 240 cells of one cyst (see text), the other metacentric lies in the center. *E* and *F*—Diakinesis illustrating the quadripartite nature of the acrocentric and metacentric bivalents and the pronounced centric constrictions of the latter: note the nucleolar remnants attached to the X chromosomes.

ential staining), is apparently absent, yet interference regularly extends in metacentric chromosomes across the centromere, so that, as in the acrocentrics, the bivalents they form possess only one chiasma[5]. It is nevertheless remarkable that in a translocation heterozygote with almost complete positive interference it breaks down, with the consequent formation of chains of three or four chromosomes[10].

From what has been said of the limitation placed by heterochromatin on the positions in which chiasmata are formed, it will be readily understandable that proof is wanting as to whether the sex chromosomes of Xy males, which appear to be totally heterochromatic, pair by means of chiasmata or otherwise. On the other hand, neo-XY pairs regularly form chiasmata between their previously autosomal segments, which, of course, are euchromatic.

In no case is positive heteropycnosis visible during the spermatogonial divisions: it develops precociously, by differential contraction, during the earliest phases of spermatogenesis.

A final anomaly occurring in the Coleoptera comprises cases of "*Callimantis*-type" meiosis[13]. In species of the carabid genus *Bembidion*, pachytene is of extreme duration and suddenly erupts into metaphase, with no evidence of the normally intervening stages having been passed through. Although the resulting metaphases have all the appearance of being composed of bivalents joined by chiasmata, there is thus no sequential evidence to support the assumption that they are in fact present.

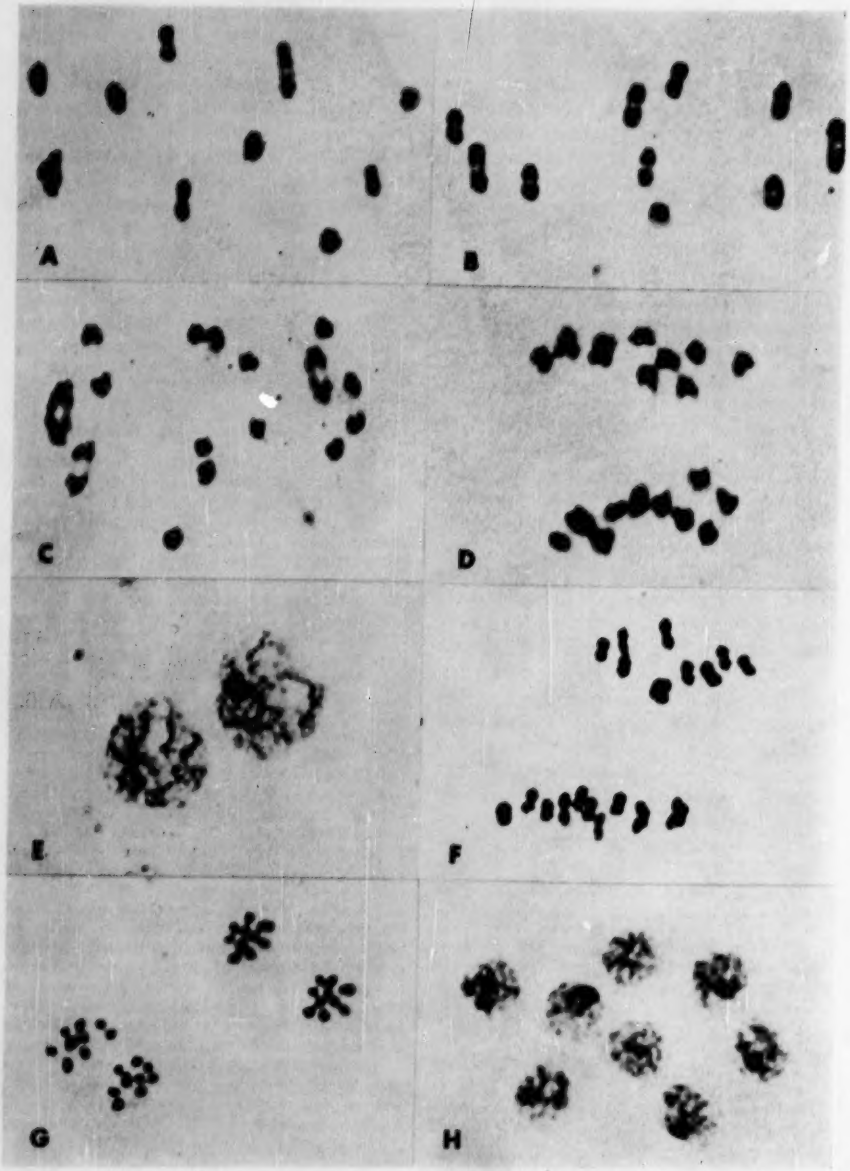
In view of this wide diversity of form and behavior in so large a group of insects, it is particularly unfortunate that relatively little is known of their formal genetics. In fact, of the species that have been studied genetically, only two in the genus *Tribolium*, *Bruchus quadrimaculatus*, and *Harmonia axyridis* have been adequately studied cytologically, although the chromosome numbers and sex-determining mechanisms of *Dermestes* spp. and *Hippodamia* spp. are recorded in the literature. As a consequence much remains to be discovered concerning the genetic significance of the more unusual modifications of the meiotic process in beetles. However, since descriptions of many of these have yet to be published, it seems appropriate at this time to give a detailed and fully illustrated account of a fairly typical and particularly well-suited species in which the chromosomes are relatively large and meiosis is diagrammatically clear. It is hoped, moreover, that the accompanying illustrations will serve, as Rhoades'[8] have undoubtedly already done for plants, a useful purpose in depicting the course of meiosis in animals.

Material and Method of Preparation

The species herein described, *Agriotes mancus* (Say), is a native North American member of the tribe Agriotini in the Elateridae, the click beetles, or wireworms as they are known as larvae. The species, like others in the genus, overwinters in the soil as adults. Spermatogenesis is initiated in the fall, suspended by the cold of winter, and resumed and rapidly completed in the spring, before or soon after emergence. It is of interest that subjection to cold storage (5°C.) halts nuclear division abruptly, so that preparations made immediately after removal from storage show all the stages of division that were formerly present.

The testes are multifollicular, closely resembling a round bunch of grapes. Following fixation for two or three minutes, the testes become opaque, thus rendering the individual follicles more easily distinguishable. It is then comparatively simple to isolate a number of follicles, preferably 12 or 16, line them up systematically in a small drop of 45 percent acetic acid on an albuminized slide, and, with heat followed by very gentle pressure, spread their contents over delimited areas one cell-layer thick (Figure 4). The individual follicles consist of a number of cysts in which the component cells are rather strictly synchronized in stage of division. As a consequence of the symmetrical alignment of the follicles and the close clustering of the contents of each cyst, the task of finding cells in a particular stage of division, usually a tedious one in "indiscriminate" squash preparations, is systematically achieved. To those accustomed to working with squashes, it is unnecessary to labor the advantages of the method.

All illustrations are from squash preparations, fixed in Smith's modification of Kahle's fluid (15 pts. 95 percent ethyl alcohol, 6 pts. formalin, and 2 pts. glacial acetic acid; freshly mixed), stained in Feulgen light green (14 mins. hydrolysis; 2 hrs. fuchsin), and photo-



PROMETAPHASE TO SECOND TELOPHASE

Figure 3

A—Prometaphase showing incomplete terminalization of the single chiasma in one metacentric bivalent and the nucleolar X chromosome (extreme right). *B*—Metaphase with per- →

graphed through a Leitz "Ortholux" microscope using B and G filters, contrast process panchromatic film, and D 11 developer. Prints were made on Kodabromide F3 and F4 paper, and with the exception of Figure 4 (× 800), are reproduced uniformly at approximately × 1825 diameters.

Description

Spermatogonia: The male of *A. mancus* has 19 chromosomes—18 autosomes and a single X (Figure 1*A*). During the series of eight spermatogonial mitoses, the chromosomes are so strongly contracted at metaphase (ranging from 1.25 to 2.5 microns in length) that the positions of the centromeres, usually marked by a constriction, are obscured. Sometimes, however, the four or five largest chromosomes are slightly flexed, which suggests, in agreement with what is seen in spermatocytes, that they are metacentric, the remainder being acrocentric. At no stage in spermatogonia is heteropycnosis evident, either positive or negative, nor is the X chromosome distinguishable from the autosomes.

Leptotene: As in most organisms, the leptotene stage of the prophase of meiosis proves to be impossible to analyze in detail. The component chromosomes are at first fine, long, slightly beaded with chromomeres, and seemingly devoid of any regular arrangement in the cell (Figure 1*B*). Soon, as the stage progresses, the X chromosome can be distinguished from the autosomes as a result of its more rapid condensation. It appears as a pancake-like body lying in close association with the nucleolus against the nuclear membrane and standing out by virtue of its stronger affinity for the Feulgen stain (Figure 1*C*).

Zygotene: By the time the chromosomes have reached zygotene, it becomes evident that their arrangement is far from being haphazard (Figure 1*D*). The ends of the autosomes are seen to be polarized against the nuclear membrane, at or near the region usually occupied by the differentially condensed X chromosome and the nucleolus, with their bodies looping out from this common point. How the chromosomes attain this orientation is as unknown for this species as it is for other organisms in which the so-called 'bouquet stage' occurs. It is probable that their initial leptotene position, unorganized though it appears to be, is actually a relic of their last premeiotic anaphase position, in which they lay with their centromeres directed towards the centrosome and their arms towards the equator. Subsequently some rapport between the chromosome ends and a particular site on the nuclear membrane must have sprung up, sufficiently strong to shift one end of the acrocentrics, at least, and to drag it to the point of polarization. This possibility suggests their possession of telomeres, end chromomeres with a special mechanical function in pairing. But if so, the telomeres are not visibly differentiated, as for example, by heteropycnosis; in fact no regions of the autosomes show the slightest tendency towards consistent differential staining.

With the start of zygotene, the pairing of homologues is initiated at the chromosome ends (Figure 1*E*) and possibly elsewhere. Judging from the relative paucity of cells with pairing unfinished, it proceeds rapidly and is completed by pachytene.

Pachytene: This is perhaps the commonest stage encountered in primary spermatocytes of this species and is the first in which the number of bivalents can be counted. Nine stout loops radiate from a common point on the nuclear membrane (Figure 1*F*). Each is composed of two chromosomes that are relationally coiled (Figure 1*G*) and intimately paired, chromomere by chromomere, throughout their length. By this time the X chromosome, still in association with the nucleolus, has undergone a profound change in shape from a pancake-like mass into a pear-shaped (Figure 1*F*) or more rarely an elongate, strap-like body (Figure 1*G*). It differs from the accompanying bivalents in its strong positive heteropycnosis and often in being polarized at one end only, so that from this point it curves out for a short distance alongside the autosomes. The autosomes, on the other hand, still fail to show even regional heteropycnosis (Figure 1*F*), despite the fact that in many Coleoptera, including the majority of elaterids examined, it is a common and especially well-developed characteristic of this stage. During pachytene the bivalents decrease markedly in length by increasing juxtaposition of adjacent chromomeres; the longest pair then measures close to 50 microns in length (Figure 1*F* and *G*). By the end of the stage the paired chromosomes show signs of becoming quadripartite through the division of each chromosome into two chromatids.

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sistent nucleolar material. *C*—Early anaphase depicting apparent ring acrocentric and metacentric bivalents following separation of their chromatids; the X chromosome is at 6 o'clock. *D*—Mid-anaphase: nine autosomes above and nine plus the X (to left) below. *E*—Two cells at interphase, one with and one without the heteropycnotic X chromosome. *F*—Second metaphase: nine autosomes in the upper cell (note parallel arrangement of the chromatids of the large metacentric); in the lower cell the oriented X lies at the extreme left and the paired chromatids of the two metacentrics are swinging into alignment between the two poles. Note especially that some of the acrocentrics in each cell show minute, bead-like short arms. *G*—Late second anaphase showing two daughter cells with nine chromosomes (to left) and two with 10 chromosomes (to right). *H*—Eight cells at telophase, four carrying and four lacking the positively heteropycnotic X chromosome.

SQUASHED CYST AT FIRST METAPHASE

Figure 4

Representative area taken from a cyst of cells at first metaphase to illustrate how with gentle pressure they spread into a layer one cell thick. Note their strict synchronization in stage of division.

Diplotene and Diakinesis: Diplotene is initiated by the four chromatids (chromonemata) of each bivalent falling apart in twos, the pair of pairs, however, remaining together, almost invariably at one point only, due to an exchange of pairing partners, i.e., due to the formation of a chiasma (Figure 1*H* and *I*). The period of transition from the pachytene bouquet shown in Figure 1*F* to the well-developed diplotene shown in Figure 1*I* is of extremely short duration: not only are intermediate stages uncommon but the definitive diplotene is reached with no appreciable change in the length of the bivalents (ca. 50 microns for the ring in Figure 1*I* and the longest bivalent in Figure 1*H*). Thereafter, however, condensation of the chromosomes is a rapid process accomplished by the paired chromonemata undergoing spiralization. At first they still retain to varying degrees vestiges of relational chromosomal coiling, which disappears quickly as the bivalents open out. They also show that the two chromonemata of each chromosome are twisted around one another (Figure 1*I*); this relational chromatid coiling is resolved more slowly but nevertheless completely by the end of diplotene.

Following opening out of the bivalents, it is readily evident that the two largest are not quadripartite throughout their entire length, for at equivalent loci in each pair a region, the centromeric constriction, has remained single (Figures 1*J* and 2*A*). In the remaining seven bivalents, the centromeres are so close to one end of the chromosomes that no visible constriction is formed, but there is no reason to believe that they differ in this respect from the metacentric ones.

In this species, unlike some other beetles, a certain simplicity of pattern is achieved by each bivalent, be it metacentric or acrocentric, as a rule forming only one chiasma (Figures 1*J*, 2*A* and *B*). What variability there is within and between cells at one and the same stage is referable by-and-large to the difference in position of centromeres and to the different positions in which chiasmata are situated: essentially all bivalents are cross shaped, deviations from equality of the four 'arms' being due to the position of the chiasma. There is, however, one major deviation from this conventional type of cell that is encountered so rarely as to appear almost as an anomaly. It consists of one bivalent assuming the form of a ring, due to the formation of a second chiasma (Figures 1*I*, 2*C* and *D*). The chromosome type involved is usually the metacentric, and the rarity of rings is attested to by the fact that after observing one in a cell at late diplotene (Figure 2*D*) a further 239 cells in the same cyst were searched without disclosing the presence of another. In other words, only one out of 480 metacentric bivalents formed a second chiasma in that particular cyst, thus setting the chiasma frequency per bivalent at a near minimum of 1.005. The efficiency of chiasma formation in this species is, however, demonstrated by the complete absence of univalents in the thousands of cells examined to date.

During diplotene the X chromosome, which had usually become pear shaped by the end of pachytene, frequently reveals that it is folded back on itself (Figures 1*J* and 2*A*). It is obviously this folding that accounts for the X alone being polarized by apparently only one end at pachytene, when actually both of its ends are so orientated but the intervening region fails to fall into a loop. Soon after the beginning of diplotene, the X chromosome loses its precocity in condensation relative to the autosomes, and its further contraction, like that of the autosomes (e.g., Figure 1*J*), is visibly accomplished by spiralization of its chromatids (Figure 2*A*). As contraction of the X proceeds, the associated nucleolus gradually diminishes in size, but as a rule remnants persist into full metaphase, by which time (Figure 3*B*), or even as early as late diakinesis (Figure 2*E* and *F*), the X is so condensed that the remaining nucleolar material is extruded from one side.

In the course of diplotene and diakinesis (Figures 1*H-J* and 2*A-F*) there is considerable movement of the chiasmata away from the centromeres (terminalization), as though these dynamic centers were already initiating the movements that end in anaphase separation. (Whether the positions in which the chiasmata were originally formed are at random or whether their more or less indiscriminate positions are a result of different rates of movement is a matter calling for extensive and detailed analysis in the future.) In general, terminalization is completed in most acrocentric bivalents by late diakinesis and in metacentrics by prometaphase.

Throughout diplotene and diakinesis, the bivalents undergo a progressive condensation. As mentioned, at late pachytene the longest measures 50 microns (Figure 1*F* and *G*); this length is maintained until very early diplotene (Figure 1*I*); shortens to 22 microns at mid-diplotene (Figure 1*J*), to about seven (Figure 2*A*) and then to about five by late diplotene (Figure 2*D*); and finally attains a minimum of ca. three microns by late diakinesis (Figure 2*F*), followed by a slight lengthening to about four microns by full metaphase (Figure 3*B*). The pachytene to diakinesis spiralization coefficient for *A. mancus*, as calculated from these measurements of the longest bivalent, is therefore 50 : 3, or approximately 17.

Prometaphase and Metaphase: Following diakinesis the bivalents become loosely oriented between the future two poles. The acrocentrics have by now assumed the form of simple rods, with a median constriction marking the position of the completely terminalized chiasma. The metacentrics as a rule are likewise rod shaped, but, besides being longer,

they have two extra constrictions that mark the positions of the two centromeres. Otherwise the chiasmata in metacentric bivalents may yet be incompletely terminalized (Figure 3*A*). Ring bivalents have not so far been detected at this stage, although they doubtless occur with an extremely low frequency.

Metaphase is marked by the definitive co-orientation of the bivalents and their congression on the equatorial plate (Figure 3*B*). A remarkable feature that these bivalents share with those of some other species of Coleoptera is the failure of the metacentrics to show a flexion at the centric regions: like acrocentrics, each metacentric bivalent lies wholly in one axis of the spindle. (This is most logically attributed to their high spiralization coefficient.) Clearly, the possibility of the 'connection' between the centromere and the centrosome being a fibre *per se* is thereby ruled out. The particular conformation of the metacentrics points more to multiple lines of force 'flowing', between the centromere and the centrosome, over the length of the free arm.

The X chromosome, now spherical or nearly spherical, usually lies just off, and to one side of, the equatorial plate. Remnants of the nucleolus are often still associated with it (Figure 3*B*) but disappear by the end of this stage.

Anaphase: Before disjunction commences, the quadruple nature of the bivalents, last clearly seen at diakinesis, is again revealed, particularly between the centromeres and the chiasma, by the separation of the two chromatids of each chromosome. As disjunction is inaugurated, this separation becomes so pronounced that the acrocentric bivalents take on the appearance of simple rings and the metacentrics are similarly modified in accordance with their basic structure (Figure 3*C*). During anaphase the poleward-moving acrocentrics are accordingly V shaped and the metacentrics Y shaped (Figure 3*D*).

The X chromosome moves off to one or the other pole just ahead of one of the daughter sets of autosomes, i.e., it segregates prereductionally (Figure 3*D*). As a consequence two numerical types of first telophase are formed: one with nine autosomes only, the other with nine autosomes and the X chromosome. Half the interphase nuclei show their possession of the X by its positive heteropycnosis, the others do not (Figure 3*E*).

Second division: Second division metaphases (Figure 3*F*) look in many ways like miniature replicas of those of the first division: they differ in the autosomes being double rather than quadruple, in the X chromosomes of those cells carrying it being barely distinguishable from the autosomes, and in the long axis of the metacentrics lying at first parallel to the equator, instead of perpendicular to it as do the acrocentrics. Before the daughter chromosomes separate, those of the metacentrics swing through an angle of 90 degrees so that all four arms become oriented on an axis between the two poles and remain touching at only one end. It is when they are aligned on the equatorial plate, and at this time only, that the acrocentric nature of the shorter chromosomes become fleetingly visible in the form of a minute 'head' at their polar ends (Figure 3*F*). From the occurrence of ring bivalents among other than the metacentric chromosomes, it is clear that these minute arms of the acrocentrics must on occasion take part in chiasma formation, but never apparently to the exclusion of the longer arms.

The division, purely mitotic in form (Figure 3*G*), gives rise to telophase nuclei either showing or not showing the X (Figure 3*H*) and thus to male-determining sperm with nine chromosomes or female-determining sperm with ten.

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GENETICS OF DDT-RESISTANCE OF AN INBRED LINE OF HOUSEFLIES

ELIZABETH THOMAS LICHTWARDT*

THE genetic study of the common housefly, *Musca domestica* L., has been until very recently almost entirely neglected. With the occurrence of resistance to DDT in the field, first reported in Italy by Sacca[16], the housefly has assumed greater importance as an experimental animal, although to date most of the genetic studies have dealt specifically with the inheritance of resistance to insecticides. Since such studies, to be worthwhile, must be based on a knowledge of basic genetics in the housefly, the work reported here was undertaken not only for the specific purpose of studying the inheritance of DDT-resistance in a laboratory strain but also to develop inbred lines which might later be useful in many types of genetic studies.

The literature to date contains diverse and often conflicting reports with respect to the inheritance of resistance in houseflies, which is to be expected in view of the variety of strains studied and the different techniques employed. DDT-resistance was reported as due to a single autosomal recessive factor by Harrison[8] and by Keiding[11]. According to LaFace[12], and D'Alessandro and Mariani[7], resistance was found to be multifactorial in inheritance. Johnson, Bogart, and Lindquist[10] obtained from their resistant strains results which they interpreted as evidence of cytoplasmic inheritance.

The work of Maelzer and Kirk[14] deserves special mention since their multiple resistant strain, "Multi X" or "Dilan", was derived from the same DDT-resistant strain, "DDT I", (sometimes referred to as D^+) which was used as foundation stock in developing the inbred resistant strain used in the research reported herein. The geneology of these strains has been reported by Bruce[2,3]. By the use of single pair crosses Maelzer and Kirk found a high resistance factor in the Multi X strain which enabled a fly to tolerate a dose up to 96 μg of DDT without mortality. This factor segregated in a 3:1 ratio, being dominant over susceptibility when 24-hour survival to a dose of eight μg DDT/fly was used as the phenotype.

In the Multi X strain Maelzer and Kirk also found a certain proportion of flies of lower resistance, some flies of this class tolerating doses up to four μg DDT/fly. By using knock-down time after both residue or drop treatment, Maelzer and Kirk suggested this lesser resistance to be the result of multiple factors.

As some significant deviations occurred in their expected ratios and since they were unable to establish a pure line of highly resistant flies from the Multi X stock, they concluded that "This highly resistant population can never be rendered homogeneous because of the low fertility associated with the high resistance factor," and inferred that flies not carrying this gene would be favored in selection over those carrying the gene.

Most of the workers studying resistance inheritance have made use of single pair progeny for testing. Many workers have used knock-down time because the limited number of flies available from

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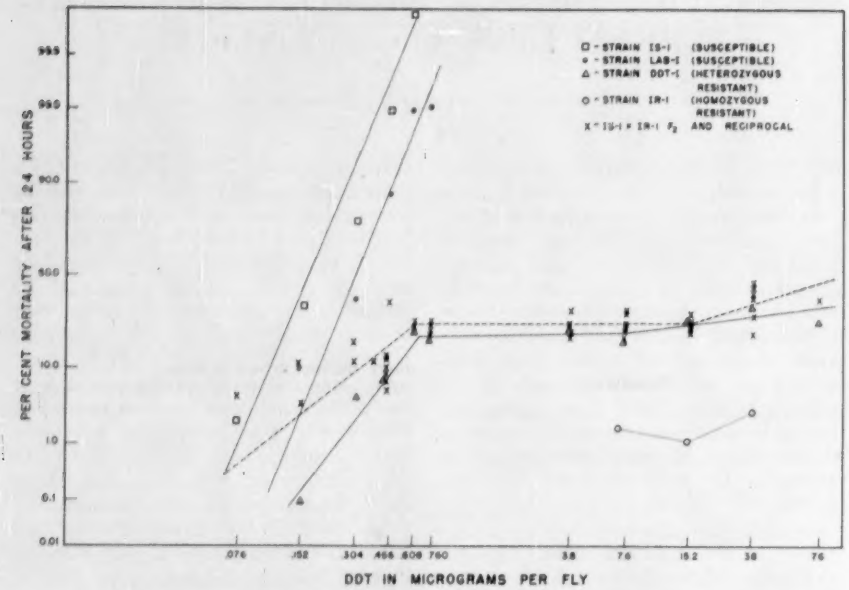

SUSCEPTIBLE AND RESISTANT STRAINS

Figure 5

Shown graphically is the DDT dosage in micrograms and the percent mortality after 24 hours for strains IS-1, Lab I, DDT I, IR-1 and reciprocal crosses.

single pairs necessitated treating the progeny identically with insecticide rather than using samples and a range of doses. It was observed in this laboratory, however, and by Busvine[5,6] and others that knock-down time is not necessarily correlated with degree of resistance to DDT in houseflies. Therefore, a genetic study by mass crossing two inbred strains, one susceptible, the other resistant to DDT was undertaken. As our phenotype for resistance we used 24-hour survival after treatment to a range of doses applied in drop form.

Materials and Methods

Strains. The two strains utilized were inbred by sibling pairs beyond 10 generations. The susceptible stock IS-1 was obtained from Dr. Bruce's Lab I strain which he selected from the NAIDM pool strain of 1948. The resistant stock IR-1 was developed from the DDT I strain which Dr. W. N. Bruce had previously selected from the Lab I strain by the use of treated larval medium and adult fly exposure to DDT. The heterogeneous Multi X strain used by Maelzer and Kirk is a multiple resistant branch of the DDT I strain, and therefore should be basically the same as IR-1 with respect to DDT resistance.

During inbreeding neither strain was subjected to any selection other than for fertility and hardiness. The techniques employed for inbreeding will be detailed in a separate paper.[13]

Breeding methods. All stages and test flies were kept at 80°F±2 and were raised according to modified NAIDM procedure[1]. Virgin females were obtained by separating emerging flies within six to eight hours.

Testing procedure. Crosses and parent strains were tested according to the method described by Dr. W. N. Bruce[4] for topical application of solutions by micro injection syringe. DDT (99 percent pure, P,P′ isomer) in acetone solution was applied in quantities of .00076 ml to the dorsal part of the thorax. Flies anesthetized by CO_2 for not longer than seven minutes were tested in samples of not larger than 60 per container, and were fed condensed milk diluted with water during the

test period. Mortality counts were taken 24 hours after treatment.

Four to seven day old females were used in obtaining most of the data for it was found that female flies gave more consistent results and were less subject to environmental effects than male flies, although males did not seem to differ in the inheritance of resistance (Table IV). Harrison[9] has made some observations on relative hardiness of male and female houseflies. By comparing results of reciprocal crosses we could test the resistance character for sex-linkage or "extranuclear" inheritance even though we used female cross progeny more extensively than males for determining resistance levels.

Results

Resistance characteristics of the inbred strains and their parent stocks

The Lab I stock tolerated doses up to .6 μg DDT/fly, whereas the inbred IS-1 strain developed from it would tolerate only up to .45 μg DDT/fly, apparently one effect of inbreeding being to decrease vigor (Table I and Figure 5). The "Canberra" strain used by Maelzer and Kirk appeared to be more susceptible than our IS-1 inbred strain, their stock giving 96 percent mortality at .192 μg DDT/fly, but they used both males and females for testing and over two and one-half times the amount of solvent which we used, factors which may have influenced their dose mortality curves.

The DDT I parent stock of the inbred IR-1 flies appeared to have a resistant fraction of about 80 percent at the dose range from .6 μg to 7.6 μg DDT/fly, after which the mortality increased slightly (Table I and Figure 5).

The IR-1 strain after 10 generations of inbreeding was almost completely pure in resistance exhibiting a slight increase in mortality at a dose of 38 μg DDT/fly (Table I). Maelzer and Kirk ascribed to their high DDT-resistance factor a tolerance of up to 96 μg DDT/fly. The ultimate dose tolerance is difficult to test in a heterogeneous population and impossible to test with single flies unless a homozygous strain is used. Although we found we could not kill a very high proportion of our resistant inbred flies with any dose of DDT topically applied, the proportion killed in crosses tended to increase slightly at or beyond a dose of 38 μg DDT/fly (Figure 5). Because of this variation in tolerance at high levels (38 μg-76 μg DDT/fly), no testing above a dose of 38 μg DDT/fly was done in most crosses.

Brief mention should be made of two genetic characteristics of the inbred strains which apparently have no connection with resistance. The IS-1 strain is pure for black abdominal sclerites, the expression of which is much more intense in the females. The inheritance of pigmented sclerites has not been studied. The IR-1 strain is pure for a single autosomal recessive factor the phenotype of which is an incomplete ("broken"=b) subcostal wing vein occurring in both wings in the two sexes. This character is not linked to the resistance exhibited in the IR-1 strain, giving a 9:3:3:1 ratio in the F_2 when crossed with the susceptible IS-1 normal wing strain.

Both inbred strains, resistant and susceptible, are relatively infertile, probably because of the effects of inbreeding, and neither strain appears to possess any distinct advantage over the other in the production of stock cultures or in crosses.

Results of crosses

Single pair crosses between the resistant and susceptible inbred strains were attempted initially to determine the degree of homozygosity of the strains. From these crosses resistance appeared to be dominant, but due to the extremely low number of fertile crosses this approach was abandoned in favor of mass crossing.

Mass crosses were carried out in three separate series. The first series was designed to compare the behavior of four substrains of IR-1 (A, B, C and D) when crossed with a single substrain of IS-1 and to determine the

TABLE I. Twenty-four hour mortality values of inbred lines, stock strains, and F_1 cross progeny females

Dose in μg of DDT/fly	Strain or Cross: Lab I stock (susceptible) no. tested	Lab I stock (susceptible) % mort.	IS-1 inbred (susceptible) no. tested	IS-1 inbred (susceptible) % mort.	DDT I stock (resistant) no. tested	DDT I stock (resistant) % mort.	IR-1 inbred (resistant) no. tested	IR-1 inbred (resistant) % mort.	S × R_{F_1}* no. tested	S × R_{F_1}* % mort.	R × S_{F_1} no. tested	R × S_{F_1} % mort.
.076	100	0.0	131	2.3								
.152	177	10.1	154	37.4	110	0.9						
.304	240	37.8	150	77.3	110	4.5						
.456	206	85.4	168	98.9	95	7.0						
.608	195	98.9	144	100.0	110	21.8						
.760	100	99.0			94	19.1						
3.8					88	22.7						
7.6					100	19.0	204	1.9	129	0.0	209	0.9
15.2					99	28.0	169	1.2			175	1.1
38.0					93	34.3	153	3.2	140	24.2	189	11.1
76.0					100	27.0						

* Hereafter the letter "S" represents flies from the IS-1 strain and the letter "R", flies from the IR-1 strain. In all diagrams of crosses female parents are always listed first, male parents last.

dose tolerance ranges. The second series was designed to clarify the discrepancies in the results of the first crosses and only one IR-1 substrain was crossed to one inbred IS-1 substrain. For the third series we utilized the same strains as for the second series with the purpose of testing one F_2 in the lower dose ranges.

Resistance as expressed in F_1 was nearly completely dominant since the tests carried out here showed that these flies were only slightly less resistant than the resistant parent strain, there being a small increase in mortality at dosage rates above 15 μg DDT/fly (Table I). There was a tendency on the part of some of the F_1 flies to knock down shortly after treatment but knock-down flies were also apt to recover and to survive for 24 hours. The F_1 tended to be more irritable than the parent strain after testing.

Cross series I. The four substrains of the IR-1 strain were crossed with one IS-1 strain (Table II). As the number of F_2 flies was limited, the dose ranges were based on the parent mortality curves, i.e., the lowest dose (.4 μg) being that which gave 98 percent mortality in the IS-1, the highest dose (38 μg) being that which gave an increased mortality in the F_1 (Table I). All possible mating combinations to the susceptible strain were attempted for all four of the resistant substrains but only five of the F_2 crosses and one backcross produced enough flies for testing.

The F_2 results indicated a single dominant autosomal factor to be producing a high degree of resistance, very likely the same factor described by Maelzer and Kirk in the Multi X strain. However, at a dose of .4 μg DDT/fly the mortality was lower than expected according to the behavior of the IS-1 parents, and the only backcross to IS-1 which produced enough flies for testing deviated significantly from the expected 1:1 ratio in favor of resistance.

Cross series II. In series II, where not more than 500 flies from any one cross were available for testing, tests were made with two doses only, .76 μg and 7.6 μg/fly. Thus attention was intentionally focused on the most reliable portion of the response curve of series I and the DDT I stock, and, furthermore, allowed a better statistical analysis well within the thresholds of the plateau of the curve.

In order to better establish the low threshold of the plateau we analyzed separately one dose below .76 μg DDT/fly. The data within the dosage rates .76 μg to 7.6 μg were grouped and analyzed as one class. As the susceptible fraction of the crosses would be apt to behave more like the Lab I stock than the inbred IS-1 we considered the 98 percent mortality level in the Lab I, i.e., .6 μg DDT/fly, as the dose which should give a clear-cut three to one segregation if no significant factors for resistance other than the single autosomal dominant were present.

Strain IR-1 (C), which was inbred for five generations beyond the first series of crosses, was mated with a second inbred IS-1 substrain for the second series. Again all possible combinations to the susceptible strain were attempted, but this time because of a sudden slump in the inbred IS-1 stock we were unable to obtain enough susceptible virgin females for a complete series of testcrosses. The three backcrosses to IS-1, however, were adequate to eliminate the possibility of any "extranuclear" inheritance, and as we had already determined by reciprocal crosses that the resistance character was autosomal we did not attempt a third series of backcrosses. Here, the results of the backcross [F_1 female (resistant female × susceptible male) × susceptible male] did not deviate significantly, as it had previously, from the expected 1:1 ratio (Table III).

Cross series III. To further study the behavior of the susceptible fraction of the crosses, the two strains used in the second series were mated (resistant female × susceptible male) and the F_2 of this cross was tested in the low dose ranges and at 7.6 μg DDT/fly. Although at one dose (.4 μg DDT/fly) the mortality was much higher than ex-

TABLE II. Twenty-four hour mortality values of F_2 and backcross females (Series I)

Cross	Dose in micrograms of DDT per fly												Probability on basis of χ^2 values
	.456		.760		1.8		7.6		15.2		38		
	no. tested	% mort.	no. tested	% mort.	no. tested	% mort.	no. tested	% mort.	no. tested	% mort.	no. tested	% mort.	.76-7.6 μg/fly
R(A) × F_{F_2}*	278	10.4	214	27.1	106	21.7	200	24.0	200	27.0	200	22.0	1.0
R(D) × S_{F_2}	200	6.0	185	25.0	200	21.0	152	30.2	141	25.5	250	10.0	.8 - .9
S × R(B)$_{F_2}$	250	14.0	200	22.5			166	30.1	250	25.6	200	46.0	.5 - .7
S × R(C)$_{F_2}$	250	7.0	249	21.6	215	25.1	250	21.0	200	26.5	202	18.0	.1 - .2
S × R(D)$_{F_2}$	200	8.5	188	21.8	200	31.0	200	24.0	200	25.5	190	45.0	.8 - .9
(R(D) × S) × S	250	42.4	297	44.7	102	47.7	250	44.0	150	47.3	173	43.0	<.01
S × (S × R(A))	91	49.0					29	65.0			50	54.0	Sample too small for analysis.
(R(D) × S) × R(D)							100	1.0	50	0.0	100	2.0	

* Symbols "A", "B", "C", and "D" designate the four substrains of the IR-1 strain.

TABLE III. Twenty-four hour mortality values of F_2 and backcross females (Series II and III)

Cross	Dose in micrograms of DDT per fly: .076 no. tested	.076 % mort.	.152 no. tested	.152 % mort.	.304 no. tested	.304 % mort.	.380 no. tested	.380 % mort.	.456 no. tested	.456 % mort.	.608 no. tested	.608 % mort.	.760 no. tested	.760 % mort.	7.6 no. tested	7.6 % mort.	15.2 no. tested	15.2 % mort.	38 no. tested	38 % mort.	76 no. tested	76 % mort.	Probability on basis of χ^2 values: .608 µg/fly	Probability on basis of χ^2 values: .76-7.6 µg/fly
SERIES II																								
$S \times RF_2$			50	4.0	50	12.0			50	12.0	126	22.2	200	22.0	200	27.5	50	30.0	50	40.0	50	38.0	.3 - .5	.9 - .95
$R \times SF_2$*													210	28.1	201	22.8								.7 - .8
$S \times (S \times R)$											105	51.4	111	47.7	133	57.1								.3 - .5
$(S \times R) \times S$													100	50.0	115	50.4								.9
$(R \times S) \times S$									150	40.6	228	50.4	190	48.9	200	45.0	150	55.3	100	42.0	50	54.0	.9	.2 - .3
$(R \times S) \times R$															100	0.0	90	5.5	97	10.3				
$(S \times R) \times R$															60	1.3								
SERIES III																								
$R \times SF_2$	100	5.0	101	10.9	103	18.4	124	12.9	133	36.0	100	26.0			117	26.4							.8 - .9	.7 - .8

* The low number available for testing here was due to an accident rather than infertility of the F_1 parents.

pected, the test at a higher dose (7.6 µg) indicated that this discrepancy might have been due to a sampling error. In the three crosses where a dose of .6 µg DDT/fly was used we found no significant deviation from the expected three resistant to one susceptible, indicating only one significant gene for DDT-resistance to be present in the inbred IR-1 strain. This autosomal dominant gene for high resistance to DDT is designated as *R*. The intermediate resistance of the Multi X strain described by Maelzer and Kirk apparently is absent in our inbred IR-1 strain. Furthermore, as the curve of the DDT I stock is so similar to that of the crosses, an intermediate form of resistance must be lacking in the resistant stock.

Discussion

The results of our genetic crosses between an inbred DDT-resistant and an inbred susceptible strain do not differ from those of Maelzer and Kirk in respect to the presence of a single autosomal dominant gene producing a high degree of resistance to DDT. However, Maelzer and Kirk suggested that this factor was reducing the fertility of the flies carrying it, the factor could not be "rendered homogeneous," and they implied that it would be selected against in a heterogeneous population. In view of the fact that our homogeneous resistant IR-1 strain was inbred beyond 10 generations by selection for fertility alone and did not appear to be at a disadvantage when crossed with its susceptible allele, we would question their interpretation of the selective advantage of the recessive allele.

Although environmental effects must influence the expression of the *R* gene, the fact that there is usually a slight increase in mortality in flies heterozygous for this gene beyond doses of 15.2 µg suggests that the gene is not completely dominant and possibly other genes modifying the expression of the *R* factor are present. On the basis of the dosage response curves, the *R* factor is the only gene in our inbred strains which initiates DDT resistance. Modifying genes would have the effect of increasing or decreasing the intensity

TABLE IV. Twenty-four hour mortality values of F_2 and backcross males

Cross	Dose in micrograms of DDT/fly: .760 no. tested	.760 % mort.	7.6 no. tested	7.6 % mort.	Probability on basis of χ^2 values: .76-7.6 µg/fly
$S \times RF_2$	127	51.1	106	27.3	<.01
$R \times SF_2$	100	26.0	112	26.7	.70 - .80
$S \times (S \times R)$	116	47.4	125	49.6	.50 - .70
$(R \times S) \times S$	90	47.7	146	59.5	.10 - .20
$(S \times R) \times R$			106	10.3	
$(R \times S) \times R$			42	4.7	

of the expression of the *R* gene rather than the production of resistance by themselves.

Because of the close relationship between the strain which we used and that used by Maelzer and Kirk we cannot generalize as to the distribution of the *R* factor in other laboratory strains or in field populations without further study, a project which we are undertaking at the moment.

Physiological genetic studies have also been initiated, and the fact that the *R* gene is the only significant resistance character present in the inbred IR-1 strain will greatly facilitate such study.

Summary

A DDT-resistant laboratory strain related to that used by Maelzer and Kirk was inbred without selection for resistance resulting in a homozygous highly resistant strain. Mass crosses between this DDT-resistant strain and an inbred susceptible strain indicated the presence, as first suggested by Maelzer and Kirk, of a single autosomal dominant gene for high resistance to DDT which was designated as *R*. No other significant resistance factors were present in this inbred strain. The *R* factor did not seem to reduce the fertility of the strain.

The inbred resistant strain IR-1 was also found to be pure for a single autosomal recessive character, a broken subcostal wing vein designated as *b* which was not linked to the *R* factor. The inbred susceptible strain IS-1 was also pure for pigmented abdominal sclerites occurring mainly in the females. The inheritance of these pigmented sclerites was not studied.

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A STUDY OF THE INHERITANCE OF CERTAIN TONGUE CHARACTERS

In 72 Pairs of Negro Twins

J. WARREN LEE*

COMPARATIVE studies between identical and non-identical twins reared together have increased our knowledge on the mechanism of inheritance of many human traits. Newman,[3, 4, 5] Rife,[6, 7] Von Verschuer,[9] and others have contributed many excellent papers and monographs on the methods of determination of identity or non-identity of twins and the mode of inheritance of many human characters through the study of twins.

The author has conducted a study of identical and nonidentical twins of school ages (six to 16) in East Baton Rouge Parish, Louisiana for the purpose of testing the mechanism of inheritance of the ability to roll the lateral edges of the tongue, the ability to fold the tip of the tongue back onto the main body of the organ, and the ability to taste phenylthiocarbamide (PTC).

As a contribution to the analysis of the mode of inheritance of tongue-rolling, tongue-folding, and tasting PTC the concordance and discordance of these characters were determined. When one member of a pair was observed to possess a certain trait, the second member was examined for the same trait. If the second member possessed the trait, the pair was considered concordant; if the second member did not exhibit the trait the pair was considered discordant.

In order to distinguish identical twins from nonidenticals, similarity tests were used. The following criteria, as used by Newman *et al.*[5] were used to ascertain whether a given set of twins was identical or nonidentical: similarity of general appearance; essential identity: height, weight, length of head, width of head, hair form, eye color, pigment pattern of iris, skin color, features of nose, lips, chin, ears, types of teeth including irregularities, type and proportions of hands and fingers. If there were a large degree of similarity between members of a pair of twins for the above mentioned characteristics, this pair was then tested for the following blood groups (ABO, Rho, rh′, rh″, and hr′) to ascertain whether a given pair of twins possessed similar blood groups and was classed identical, or had dissimilar blood groups and was classed nonidentical. Of the original 26 pairs of probable identical twins determined on the basis of the similarity tests, only four pairs of twins were rejected because of blood group incompatibilities.

Data in Table I show the distribution of identical twins, nonidentical twins (like sex) and nonidentical twins (unlike sex). These frequencies are not significantly different from expectation for Negro twins in the United States in general.

Table II shows the mean differences in height, weight, head length and head width between identical twins, nonidentical twins (like sex) and nonidentical twins (unlike sex).

Table III shows the concordance and discordance for tongue-rolling, tongue-folding and the ability to taste PTC in 72 pairs of identical twins, nonidentical twins (like sex), and nonidentical twins (unlike sex).

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Discussion

The identical twins had one pair (Roy and Ralph) to show discordance in two traits (rolling, tasting); the nonidentical twins of like sex had a single case (Willie and Emmett) showing discordance in tongue-rolling and ability to taste PTC; and the nonidentical twins of unlike sex had two pairs of twins to show discordance in two traits: Lillie and LeRoy exhibited discordance in tongue-folding and the ability to taste PTC; and Eddie and Shirley were discordant in tongue-rolling and the ability to taste phenylthiocarbamide.

The identical twins exhibited a smaller mean difference between members of a pair for height, length of head, and width of head than the nonidentical twins. This closeness in the above measurements between identical twins living together should be expected since they have the same genetic constitution and have been exposed to similar environmental conditions. On the other hand, mean differences for height, weight, length of head, width of head between members of nonidentical pairs were about twice that of the identical pairs. This merely indicates that members of nonidentical pairs had differed in genetic constitutions from one another.

Sturtevant[5] and Hsu[2] have suggested that the ability to roll the lateral edges of the tongue is due to a dominant gene, while the inability to roll the lateral edges of the tongue is recessive. One set of the 22 sets of identical twins observed was discordant. Twenty-one pairs of identical twins were concordant. The discordance in identical twins is considered to be a measure of environmental influence upon certain traits. Concordance is considered a measure of the hereditary influence upon a trait. Among the nonidentical twins, 20 sets of the 50 studied exhibited discordance for at least one of the three characters studied. This high degree of discordance may be due to the presence of different genes in members of each pair of the nonidentical twins.

Hsu[2] has indicated that the ability to fold the tongue is inherited as a recessive trait, and the inability to fold the tip of the tongue is dominant. It was observed that one pair of identical twins was discordant, and 21 pairs of identical twins were concordant for tongue folding. The data (Table III) indicate that heredity exercises a much greater influence on folding than environment. There were four pairs of nonidentical twins showing discordance, and 46 pairs were observed to show concordance for the tongue folding character.

Ardashikov *et al.*,[1] in a study of the diagnosis of twins, found three pairs of identical twins out of a total of 137 differed in their ability to taste PTC. The results of this study indicate that two pairs of identical twins differed in their ability to taste PTC, and 20 pairs of identical twins showed concordance for taste acuity to phenylthiocarbamide. There were nine pairs of nonidentical twins showing discordance, and 41 pairs were observed to show concordance for the taste acuity to phenylthiocarbamide.

It will be necessary on considerations of the genetics of tongue-rolling, folding, and taste ability to take into account the facts reported on discordance and concordance of twins.

Summary

Seventy-two pairs of Negro twins of school age (six to 16) in East Baton Rouge Parish were studied. Of this number, 22 pairs were identical, 30 pairs were nonidentical of like sex, and 18 pairs were nonidentical of unlike sex.

The nonidentical twins studied exhibited mean differences for height, weight, head length, and head width slightly more than twice the differences in the identical twins (Table II).

Data from the study indicate that the identical twins possessed a concordance of 95.5 percent for tongue-rolling, whereas the nonidentical twins of like sex had a concordance of

TABLE I. Distribution of Negro twins of school age in East Baton Rouge Parish

Identical		Nonidentical (Like Sex)		Nonidentical (Unlike Sex)	
Actual Number	Percent	Actual Number	Percent	Actual Number	Percent
22	30.6	32	44.4	18	25.0

TABLE II. Average differences between the two members of identical twins, nonidentical twins (like sex) and nonidentical twins (unlike sex)

Differences	Identical	Nonidentical (Like Sex)	Nonidentical (Unlike Sex)
Height cm	2.0	5.2	5.7
Weight lbs	1.4	7.6	8.8
Head length mm	8.4	17.3	18.7
Head Width mm	5.8	11.8	12.5

TABLE III. Concordance in Negro twins of school age in East Baton Rouge Parish

Concordance (++) and Discordance (+−) for Tongue-rolling, Tongue-folding, and the Ability to Taste Phenylthiocarbamide in 72 Pairs of Negro Twins

		Percent		Number
		++	+−	of pairs
Tongue-rolling	Identical	95.5	4.5	22
	Nonidentical (Like Sex)	84.4	15.6	32
	Nonidentical (Unlike Sex)	88.9	11.1	18
Tongue-folding	Identical	95.5	4.5	22
	Nonidentical (Like Sex)	90.7	9.3	32
	Nonidentical (Unlike Sex)	77.8	22.2	18
Tasting (PTC)	Identical	91.0	9.0	22
	Nonidentical (Like Sex)	84.4	15.6	32
	Nonidentical (Unlike Sex)	77.8	22.2	18

only 84.4 percent and nonidentical twins of unlike sex had a concordance of 88.9 percent.

The identical twins demonstrated a concordance of 95.5 percent for tongue-folding, the nonidentical ones of like sex had a concordance of 90.7, and the nonidentical twins of unlike sex had a concordance of 88.9 percent for the same trait.

Monozygotic twins exhibited a concordance of 90.9 percent for the ability to taste PTC, while the nonidentical twins of like sex had a concordance of 84.4 and nonidentical twins of unlike sex had a concordance of 77.8 percent for the same trait.

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TRANSLOCATION IN NARANJILLA, AN ECUADORIAN SOLANACEOUS FRUIT

Patricia Sarvella*

SOLANUM quitoënse Lam. is known to the natives of the Ecuadorian Andes as Naranjilla, Spanish for small orange, because of its small round fruit. Chalons[1] reported that the flavor of the juice has the tartness of a mixture of orange, pineapple, and tomato juice. A young Naranjilla plant is illustrated in Figure 6*A*, and Figure 6*B* shows flowers and young fruits. Taxonomically *S. quitoënse* has been placed by De Candolle in the same subsection of the genus *Solanum* as the eggplant.

Recently attempts have been made to introduce Naranjilla into North Carolina as a greenhouse crop. Poor fruit production led to a study of the nutritional requirements of the plant.[3] Pollen examination of various plants showed them to be partly sterile with 10 to 70 percent empty grains. Therefore a cytological study was made of mitosis and meiosis to determine if chromosomal abnormalities were the basis of this sterility.

Cytology

Aceto-carmine squashes of tapetal cells of young anthers showed a somatic chromosome number of $2n = 24$ (Figure 7*A*), which was the number reported by McCann[2] in root tip cells.

Pollen mother cells were examined from five plants grown from seeds of Ecuadorian market fruits. Four of the five plants had irregular chromosome behavior. Only one plant regularly formed 12 bivalents at metaphase (14 out of 15 PMC's), yet it produced some sterile pollen. A second plant had a much lower frequency of bivalents, namely a modal number of 11 bivalents and two univalents in nine out of 18 PMC's with a maximum of eight univalents in one cell. The remaining three plants showed a ring of four conjugated chromosomes indicating an interchange of chromosome segments. A typical ring is shown in Figure 7*B*. At least two different transloca-

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THE NARANJILLA PLANT

Figure 6

A—Growth habit of a young Naranjilla plant; *B*—flowers and young fruits.

NARANJILLA CHROMOSOMES

Figure 7

A—Somatic chromosomes of Naranjilla (ca 1300 ×); *B*—metaphase I. Pollen mother cell showing 10 bivalents and a ring of four. (Detritus below second bivalent from left).

tions seemed to occur since in plant 1 the ring appeared to be attached to the nucleolus while in plants 2 and 7 it was not. Also the ring of four occurred less frequently in plant 1 than in plants 2 and 7 (Table I). Conclusive proof of the presence of two different translocations might have been obtained by crossing plant 1 with plants 2 and 7 but unfortunately plant 1 died before the crosses could be made.

Future Possibilities

These preliminary studies indicate the occurrence of structural heterozygosity in Naranjilla. This variability could be used to improve the species and to adapt it to non-Ecuadorian conditions. Further work is required to establish the relationship between the chromosomal interchanges and pollen sterility, and to determine the extent and nature of the interchanges in natural Naranjilla populations.

TABLE I. Frequency of interchanges in plants with translocations

Plant Number	Number of PMC's with		
	Quadrivalent	Trivalent	No multivalents
1	1	4	14
2	25	0	1
7	15	2	1

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PUPAL LETHALS IN *DROSOPHILA MELANOGASTER*

FRANK SETO*

THE relatively small amount of descriptive information concerning *Drosophila* lethals despite numerous reports in genetic literature was commented on by Hadorn[9] in a review article. Since then studies have been made on the times of action of several groups of lethals by different investigators[10, 16, 17, 20], but these studies did not describe in detail the pattern of damage expressed by the mutants. The author has selected several second-chromosome "pupal" lethals collected in the course of an earlier study[20], and others isolated subsequently, in order to describe their pattern of damage. This paper summarizes the results of observations made on the gross morphology of these lethals.

Materials and Methods

The second-chromosome pupal lethals were extracted by means of the "sifter" technique[15] and kept in a balanced condition with *Cy cn*^{2} *L*^{4} *sp*^{2}. The origins of the different lethals are listed in Table I. Shortly after the lethals were collected, they were tested for allelism by crossing the balanced stocks with one another.

Observations of metamorphosis in normal flies (*Mass 38, W'is 53*) were made so as to become familiar with the normal processes, knowing which, one can better evaluate the deviations displayed by the lethals. Many of the observations were made on living specimens but in order to study certain parts in detail it was necessary to supplement the live examinations with dissections. Before the main part of the investigations was begun, the relative constancy of mutant expression was ascertained by outcrossing the stocks to wild (*W'is 53*) and *cn bw* (cinnabar eye, brown eye) strains, and comparing the resulting phenotypes in these genetic backgrounds with those expressed in the balanced stock. The characteristic features of the lethals were present in each of the three backgrounds, although they varied in minor details. The following observations and descriptions are based on the study made in one background, that of the balanced stock.

Each lethal was observed with respect to puparium formation and externally visible changes. Certain changes in metamorphosis, such as the transformation of the third instar larval skin into the puparium, the eversion of the appendages (wings, legs, and cephalic complex), the pigmentation of the eyes and certain hypodermal derivatives, the dissolution of the fat bodies, and the shaping of the body into head, thorax, and abdomen can be seen readily in the fresh undissected specimens. The prepupa, whenever it could be freed from the puparium, and the pupa were examined in Drosophila Ringer's solution with a dissecting microscope (at 10×, 30×, and 60×). Some were fixed at various ages and stored in alcohol for later examination. Others were mounted in balsam or Hoyer's—the latter a medium which facilitates the study of the exoskeleton.

Observations

Metamorphosis in Drosophila

Table I which summarizes briefly the more conspicuous changes that take place in the post-embryonic development of *Drosophila* is presented for comparison. Detailed descriptions of metamorphosis may be found in papers by Robertson[19] and Bodenstein[1].

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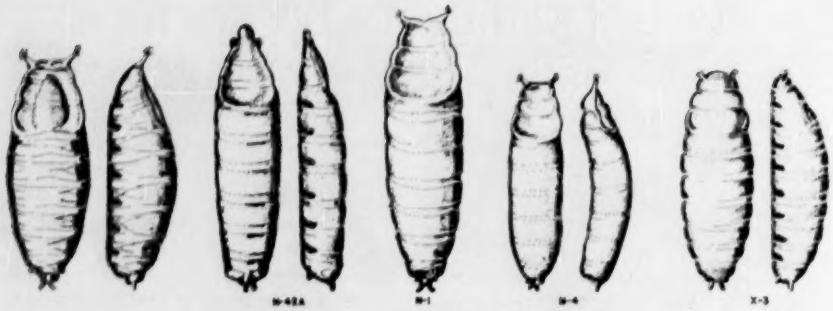


NORMAL AND LETHAL PUPARIA

Figure 8

The dorsal and left lateral views of the normal are shown on the extreme left. Lethal puparia are illustrated as N-42A, N-1, N-4, and X-3.

Cessation of development and pattern of damage

Preliminary observations were carried out to determine the approximate time or times in pupal development when the lethal manifested itself. In this study, the late post-embryonic development was partitioned into the following intervals: larval (L), prepupal (Pr), early pupal (EP), late pupal (LP), and adult (A). One thousand eggs (10 relicates of 100 eggs) for each of several mutants were started out in vial cultures, and the extent of development observed for those surviving beyond the third instar larval stage. With a 10 percent correction for miscellaneous inviability, from the balanced lethal mating scheme one would expect about 450 non-lethal (NL) individuals to emerge as adults. The proportion of lethals should be about one-half of this or 225. The results of this study are summarized in Table III.

TABLE I. Normal development of *Drosophila melanogaster* at 25°C

Hours	Post-embryonic events
0	Larva hatches from egg
25	*First larval molt*
48	*Second larval molt*
96	Larva leaves food to pupate; becomes quiescent
	Formation of puparium
	Larva shortens, broadens to assume ellipsoid shape; first segment retracts; anterior spiracles evert; contraction ceases; opercular seams apparent; puparium yellowish
98	Larval segmentation becomes indistinct; shaping for puparium complete; fully colored
100	*Prepupal molt*
	Body contracts in waves; prepupa retracts from the larval skin (puparium); anterior and posterior tracheal trunks discarded; mouth hooks ejected
104	Gas bubble in abdomen increases in size; wing and leg sacs evert, the latter elongating; dissolution of fat bodies
108	Frontal sacs evert
	Pupal molt and beginning of pupation
	Body constricts into head, thorax, and abdomen; still colorless; wings and legs continue to enlarge; fat body fragments floating in the hemolymph
145	Shaping of body completed; brown eye pigment develops; wings begin to fold
165	Scarlet eye pigment produced giving wild-type eye color; head and thoracic bristles become pigmented
178	Rest of body becomes pigmented
192	*Differentiation complete; adult ready to emerge.*

The lethals studied do not all necessarily display their main effects during the period of pupation. Lethals with effects in the larval or the adult stage have been included to show the possible range of expression manifested by the lethals as a group. Although the phenotypes can be altered somewhat by changes in cultural conditions and differences in genetic backgrounds, the essential features of each lethal remain the same. Moreover, several of the lethals may cease development at more than one period in pupation and appear to have different patterns of damage. With this in mind the following brief descriptions attempt to give only the more characteristic features of the lethals.

Of the 15 lethals studied, only five showed marked deviations in puparium formation. The puparia of four of these are shown in Figure 8. Following are brief descriptions of the abnormal puparia:

N-42 (Figure 8). Generally longer and greater in mass; shape larva-like; body segmented and elongated with slight dorsal-ventral flattening, first larval segment unretracted and anterior spiracles uneverted. Color of warmer hue but less intense than normal; pigment uniformly distributed or concentrated at the joints. Exoskeleton thinner, fragile and often collapsed.

N-1 (Figure 8). Longer and slightly larger; shape varies from larva-like forms to those hardly distinguishable from normal; most, intermediate in shape. Color and texture of exoskeleton normal.

N-4 (Figure 8). Usually smaller and more slender than normal; about one-third the normal mass. Shape elongate with tendency towards concavity on ventral side; opercular seams may or may not be present. Early dying forms at first colorless but becoming irregularly discolored with age. Exoskeleton usually thin and fragile. Puparia of late dying forms more nearly normal.

X-3 (Figure 8). Somewhat smaller and dumpier than normal.

N-61. Normal-appearing except for the texture and color of the exoskeleton. Color dark, deepening to almost black with age; exoskeleton thicker and more resistant.

The "pupal" lethals have been classified into two groups according to the extent of their development. If the mutant died before the cephalic complex was everted it was classified as a larval-prepupal lethal; otherwise it was considered a pupal lethal.

Lethals which express their effects in the larval or prepupal stages

N-42A (Figure 9). Anterior part of prepupa adheres to puparium; posterior end free and contracted away. Space in between filled with a light-colored fluid, often oily in older specimens and containing particles of the disintegrated fat bodies. Prepupal cuticle present but incomplete, broken where it adheres to the case. Posterior tracheal trunks discarded, anterior trunks and mouth hooks still attached to body. Wing, leg, and frontal sacs undeveloped.

X-3 (Figure 9). Prepupa deformed and only partly filling puparium. Except for an irregular knoll of tissue on the anterior end (vestiges of head and thoracic structures), body sac-like. Prepupal cuticle intact, body colorless except for small specks of melanization of the anterior knob. Pigmented patches represents melanotic degeneration of larval tracheal trunks. Wing, leg, and frontal sacs undeveloped or absent.

N-61 (Figure 9) and *N-51.* Anterior end retracted from case, anterior and posterior tracheal trunks ejected, mouth hooks still attached to body. Body distinctly separated into anterior (cephalothoracic) and posterior (abdominal) regions by incomplete band of sclerotized tissue. Prepupal cuticle intact posteriorly, broken anteriorly allowing free-floating fat body fragments to escape into the fluid filling the space between the prepupa and puparium. Often large gas bubble present within the body. Wing and leg sacs everted, development limited. Frontal sacs uneverted, eye rudiments in cryptocephalic position often pigmented.

N-51. Develops further than *N-61;* puparium formation unaffected.

N-1 (Figure 9). Anterior end often retracted, tracheal trunk discarded, mouth hooks often attached to body. Body indistinctly separated into anterior and posterior portions. Wing, leg, and frontal sacs slightly developed, rarely everted. Eye rudiments pigmented in exceptional individuals and appearing as two dark red bodies in their cryptocephalic position. Strain lost.

N-59. Prepupal cuticle complete and intact. Prepupa separated from case, tracheal trunks and mouth hooks remaining attached to body. Incomplete band of sclerotized tissue separating anterior and posterior regions; body otherwise shapeless. Wing and leg sacs infrequently everted but undeveloped. Frontal sacs undeveloped. Note: death predominantly in the larval stage; penetrance incomplete. Strain lost.

X-11. Melanotic degeneration of larval tracheal trunks which causes adhesion of prepupa to puparium; otherwise similar to the normal prepupa in morphology. Cephalic complex rarely everted; wings and legs variably developed. Strain lost.

Lethals which express their effects mainly in the pupal stage; occasionally surviving as sterile adults.

N-50 (Figure 9). Wing, leg, and frontal sacs everted (phanerocephalic condition); wing and leg sacs elongate but development limited. Body colorless except for melanotic

TABLE II. Source of pupal lethals

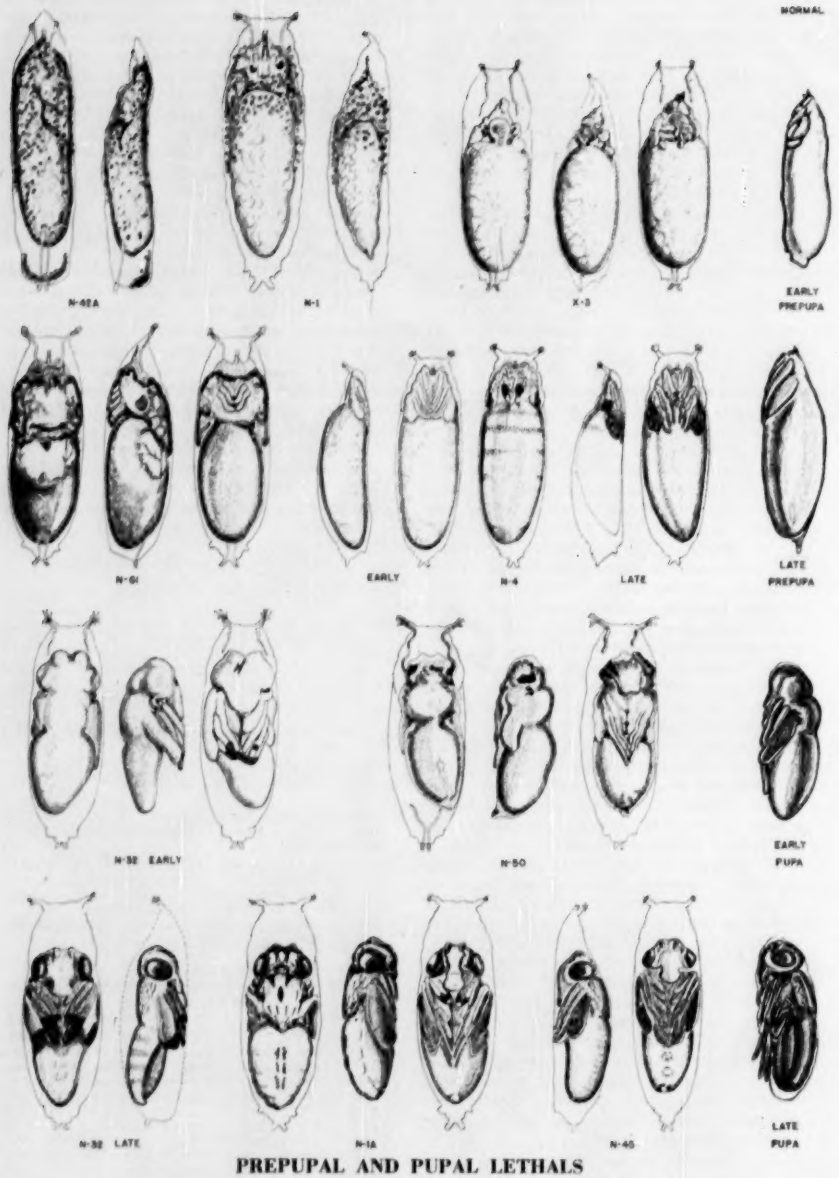
Origin	Lethal stock	Designation
Collected from natural population; Madison, Wis. 1951	1(2)Mad 5 l^{n-1a}	*N-1*
	1(2)Mad 5 l^{n-4}	*N-4*
	1(2)Mad 5 l^{n-32}	*N-32*
	1(2)Mad 5 l^{n-42}†	*N-42*
	1(2)Mad 5 l^{n-42a}	*N-42A*
	1(2)Mad 5 l^{n-50}	*N-50*
	1(2)Mad 5 l^{n-51}	*N-51*
	1(2)Mad 5 l^{n-59}*	*N-59*
	1(2)Mad 5 l^{n-61}	*N-61*
Collected from natural population; Madison, Wis. 1953	1(2)Mad 5 l^{n-1a}	*N-1A*
	1(2)Mad 5 l^{n-45}	*N-45*
Induced in wild type stock *Mass 38* with ca 2000 *r* of X ray 1951	1(2)Mass 38 l^{x-6}	*X-3*
	1(2)Mass 38 l^{x-11}*	*X-11*
Induced in *cn bw* stock with ca 1700 *r* Co-60 1951	1(2)cn bw^{co-3a}	*Co-3A*
	1(2)cn bw^{co-7}	*Co-7*

* Stocks lost in the course of experimentation.
† *N-42* and *N-42a* with different manifestations derived from a single mutant. *N-42* subsequently lost.

TABLE III. Specific times of manifestation of pupal lethals

Stock	Lethality						Non-lethals Total
	Larval	Pre-pupa	Early Pupa	Late Pupa	Adult	Total	
N-42A	43	142	....	....	....	142	409
X-3	1	115	....	....	....	116	447
N-61	5	155	....	....	....	158	407
N-51	....	205	....	....	....	205	315
N-4	(20)*	29	83	46	....	158	415
N-50	....	(14)	162	....	....	162	423
N-32	....	(14)	133	60	1	194	404
N-1A	....	(1)	3	210	....	213	426
Co-7	....	(13)	131	70	1	202	467
Co-3A	(2)	(13)	100	71	16	187	420
N-45	(13)	(8)	14	86	20	110	389

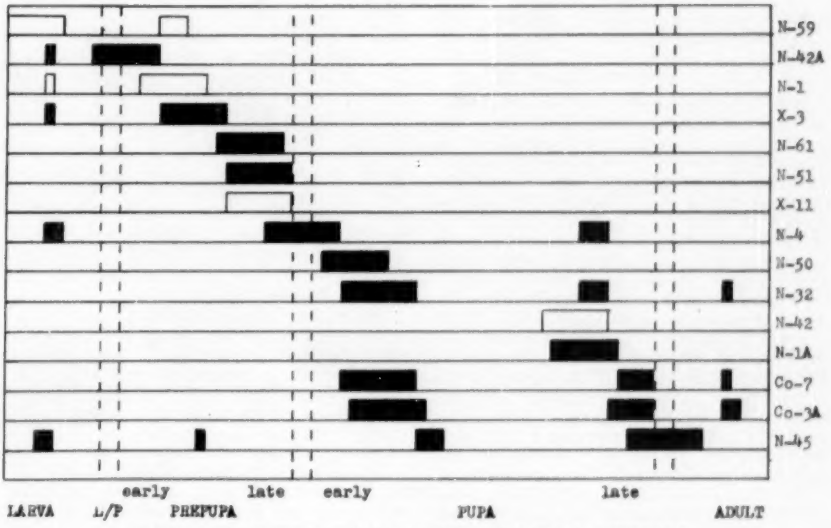
* Numbers in brackets represent unidentifiable individuals.



PREPUPAL AND PUPAL LETHALS

Figure 9

At the end of each row is a simplified drawing showing the left lateral view of a normal individual of approximately the same stage of development. Top row—early prepupal lethals; second row—late prepupal lethals; third row—early pupal lethals; bottom row—late pupal lethals.



TIME OF ACTION OF A SERIES OF PUPAL LETHALS

Figure 10

The positions of the bars give the approximate times when cessation of development and subsequent mortality occurs. The longer bar indicates the period of greatest effect, the shorter bar, another effective period and the short vertical line, occasional manifestations either in the larval or imaginal stages.

degeneration of hypodermis in regions of compound eyes, tip of abdomen, and often the leg joints. Eye pigmented in exceptional individuals. Pupal cuticle often broken, fat body fragments escaping into fluid outside pupa.

N-32 (Figure 9). Some cease development early, others late. Early dying form: similar to *N-50* but without the characteristic distribution of pigment; discoloration limited usually to leg joints, tarsal segments, and occasionally the wing tips. Pupal leg and wing sheaths often adhere to puparium and do not elongate. Legs develop in cramped position. Water-retaining capacity of cuticle affected, and pupa becomes badly shrunken. Under optimum conditions lethal may differentiate fully—eye and hypodermis pigmented, hairs and bristles developed. Exceptional individuals emerge as weak and sterile adults.

N-4 (Figure 9). Both early-dying and late-dying forms *cryptocephalic*, i.e., cephalic complex uneverted and retained within the thorax. In early acting forms the prepupa or pupa colorless; wing and leg sacs everted, differentiation limited. In late-dying forms differentiation of externally visible parts variable: development not as complete posteriorly. The eyes pigmented, legs hairy and pigmented as are the wings. The phenotype is similar to the *cryptocephal* (*crc*) lethal described by Hadorn and Gloor[11].

N-1A (Figure 9). Structurally, differentiation almost complete. Formation and disposition of melanoid pigment abnormal: lethal pupa at first slightly off shade, later becoming dark brown with patches of heavy melanization in thicker parts of the exoskeleton. In older specimens nephrocytes deeply pigmented and conspicuous.

Co-3A and *Co-7*. Phenotypes alike. Early-acting forms similar to *N-32*. Late-dying forms often completely differentiated; infrequent imagines usually weak and generally sterile.

N-42. With exception of irregular pigmentation of compound eye, differentiation of external parts nearly complete. Head somewhat transparent; filled with hemolymph and fat body fragments; brain degenerated or poorly developed. Strain lost.

N-45 (Figure 9). Body slightly smaller and more slender. Sheaths about legs do not elongate; legs develop in this cramped position; become stunted and twisted. Under optimum conditions, many reach the point of emerging. Most of these fail to break free; die partly emerged from the puparium.

Those emerging successfully generally weak and unproductive.

Discussion

Each lethal factor displays a characteristic pattern of damage associated with cessation of development. Although the spectrum of effects can be altered by changing the cultural conditions or by introducing genetic modifiers, the phenotype of each lethal is still distinctive enough in spite of its apparent variable expression. The lethal factors, taken as a group, may affect puparium formation, reduce larval and imaginal viability, decrease reproductive capacity as well as stop development in the larval, prepupal, or pupal stages.

From the pattern of damage associated with the cessation of development, the times when the lethal effects manifest themselves can be approximated. These are summarized graphically in Figure 10. Although the times of mortality seem to be distributed throughout the "pupation" period, with the possible exception of two (*N-4* and *N-45*) the lethal expression of a particular lethal appears either in the larval-prepupal or the pupal interval. Seven of the fourteen lethals show their effects predominantly in the larval-prepupal stage and rarely later. The larval and prepupal phases may be thought of as parts of a single stage, since the latter is an extension of the larval period of life. Snodgrass[12] as to *Rhagoletis*, Fraenkel[4] as to *Calliphora*, and others have shown that the prepupa is a quiescent fourth larval instar and the formation of the pupa occurs shortly after the eversion of the frontal sacs and the pupal molt. In the higher Diptera, modifications in pupation involve not only a quiescence of the fourth larval instar (prepupa) which precedes the pupal instar, but also the change of the molted skin of the third larval instar into a protective covering (puparium) which houses the transforming immature fly. It is therefore suggested that the apparent "pleiotropism" associated with prepupal lethality, such as larval mortality, abnormal puparium, the failure in development of imaginal structures, and cessation of development during the *L/P* and *Pr* periods can be defined as extended manifestations of the larval pattern of damage.

The other mutants show little lethality in the larval-prepupal stage, but die as pupae; and in the case of four of these, weak and unproductive imagines emerge. Although *N-32*, *Co-3A*, and *Co-7* cease development either in the early part of pupation or later, the mortality is still restricted to the pupal stage proper. The fact that one group of lethals fails to develop beyond the prepupal stage whereas the other continues beyond the pupal molt only to die later, suggests further that the prepupal and pupal stages are distinct, although both occur in the puparium and play a part in the metamorphosis of *Drosophila*.

With the possible exceptions of *N-4*, in which both prepupal and pupal deaths occur, and of *N-45*, which displays a general viability effect affecting all stages, the mutants appear to be *phase-specific*. The results of this preliminary morphological study are consistent with Hadorn's concept of phase specificity[7], in that the lethal manifestations of any one lethal gene are limited to some particular stage of development, rather than occurring continuously throughout the post-embryonic development. However, a morphological study of this type must be supplemented with an experimental approach before the initial time of action and origin of the pattern of damage can be elucidated.

A number of pupal lethals have been described in the literature; most of these casually, but others in great detail. Some of these include *Pale deficiency*[13], *lethal giant*[6], *bloated larva*[3], and *S-84*(*D. willistoni*)[18], which have their lethal effects in the larval-prepupal interval; and *lethal translucida*[8], *29P*(*D. willistoni*)[18], *head defects*[14], *cryptocephal*[11], *triplo-X superfemale*[13], *eyegone*[12], *engrailed*(*D. hydei*)[22], *focal melanosis*[5], and *1(3)e 3-In(3R) C*[2], which have been described as having their effects in the pupal interval.

Summary

A series of 15 "pupal" lethals was investigated to determine more closely their time of manifestation within the pupal stage. The preliminary study of their patterns of damage included observations of puparium formation and externally visible changes of the prepupa and pupa during this period. The lethals appear to fall into two groups according to the extent of development. Seven of the 15 lethals failed to develop beyond the prepupal stage, and some displayed larval mortality and/or abnormal puparia. The others displayed little prepupal mortality, ceased development mainly as pupae, and some even gave rise to occasional sterile adults. With accountable exceptions, the lethal effects were characteristic for each stock and *phase-specific*, i.e. they occurred at specific stages in development.

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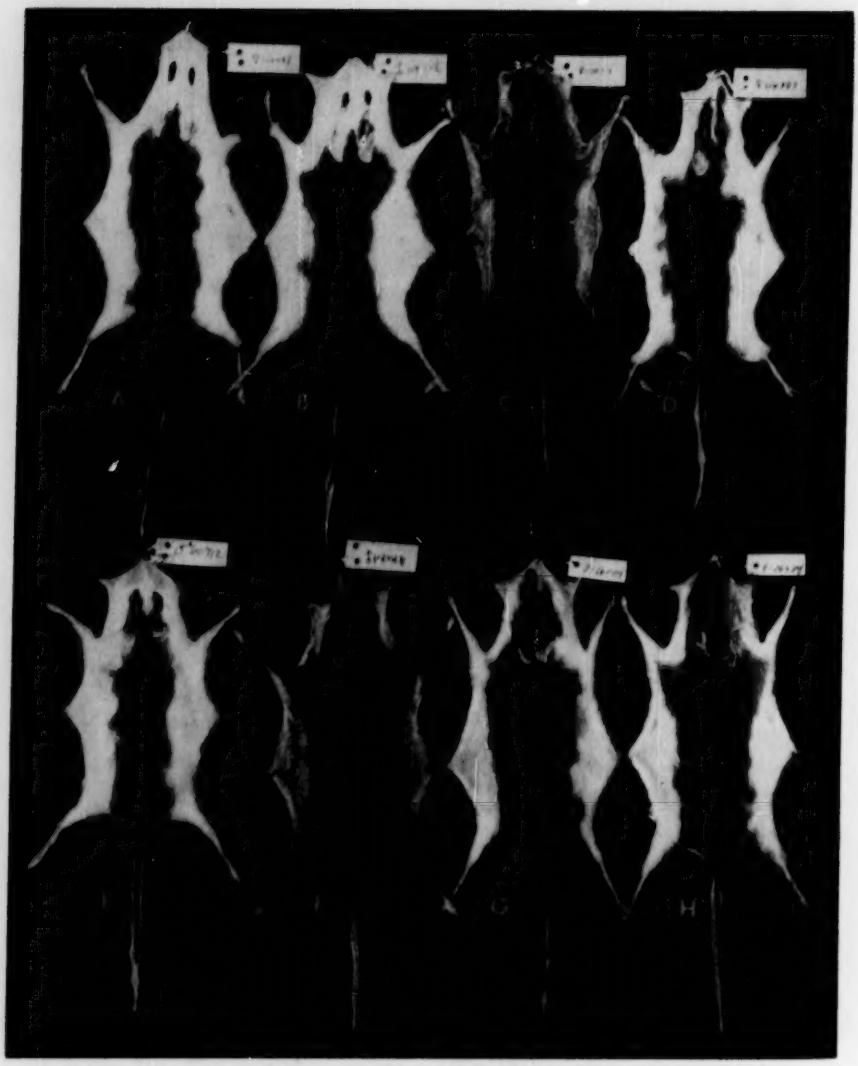
A NEW PUBLICATION POLICY

During the past few years the financial strain of publishing manuscripts of considerable length has become great. In the last 10 years the cost of both printing and engraving has risen appreciably, with little increase in income. Except in a few exceptional cases, it has not been our policy in the past, to ask financial assistance from contributors to the *Journal*. We regret that production costs make it necessary at this time to change that policy.

At a meeting of the Council of the American Genetic Association, held on January 19, 1956 it was unanimously voted that as of January, 1956 all manuscripts accepted will be limited to eight pages, including illustrations, without a surcharge. The author of an article exceeding this limit will be charged at the rate of $20.00 per page for each additional page. Contributors will also be asked to help defray the cost of excessive numbers of illustrations.

—Barbara Lake
Managing Editor



VARIATION IN WHITESIDED PEROMYSCUS

Figure 11

A and *B*—High-grade whitesides; *C*—wild-type sib of *E* below; *D*—whiteside, note ragged lateral line; *E*—one of the original whitesides and source of the majority of whiteside genes in the stock; *F*—another wild-type mouse; *G* and *H*—low-grade whitesides.

WHITESIDE, A NEW MUTATION IN PEROMYSCUS

WILLIAM B. MCINTOSH*

AN undescribed mutation, here named whiteside, has appeared among the *Peromyscus maniculatus* maintained at the Laboratory of Vertebrate Biology. The mutation first appeared in the offspring of a mating of F_2 mice from a cross between stocks of *P.m. bairdi* and *P.m. gracilis*, both from Michigan. A female mated to a double first cousin bore litters of three and five young. One male in each litter was a whiteside.

The *gracilis* stock had been maintained in the laboratory for several generations before the original subspecific cross was made. The *bairdi* stock had recently been taken in the field. Several generations have been reared in each stock since the discovery of the mutant, and a fair amount of inbreeding has occurred. Nevertheless, the mutant has not appeared in either of the parent stocks. It thus appears possible that the mutation actually originated in the laboratory in one of the animals involved in the interracial cross.

Description

The most prominent feature of whitesided mice is the great amount of white in the pelage. In wild-type *Peromyscus* ventral hairs are devoid of pigment in their distal portion, and are heavily pigmented in the proximal part. Depending on the length of the unpigmented portion of the hairs, the over-all color of the ventrum varies from grayish to white. Small areas in which the hairs are entirely white are relatively common among normal *Peromyscus*, but these spots rarely occupy any extensive area. In wild-type individuals there is a rather sharp transition at the lateral line between the white-tipped ventral hairs and the colored lateral and dorsal ones. When viewed from above, the white underparts of such mice are scarcely visible.

Whitesided mice have the entire ventral surface covered with completely-white hairs. These all-white hairs are indistinguishable from hairs of albino *Peromyscus*, and they extend well up onto the sides of the body, in effect moving the lateral line dorsad by perhaps half a centimeter. The lateral line in whitesides is usually ragged in outline, whereas it is reasonably straight in normal *Peromyscus*. It is this dorsad extension of the white areas which give the mutant its name.

The top of the head is white to varying degrees. In only two cases have we observed a whiteside which had no trace of white on its head. In a few other cases whitesided mice had only very small areas of white here (see Figure 11*G*). Characteristically there is at least a narrow stripe through the middle of the forehead, and in some individuals the entire head is white. The skin and hairs of the ears are also more or less white, with the degree of whiteness roughly correlated with the amount of white on the head. Even the two whitesides which had no white hairs on the top of the head had ears which were paler than those of normal mice. The tip of the tail usually had some white on the dorsal surface. The characteristic range of variability may be seen in Figure 11.

In the whiteside strain all mice possess the gene for yellow agouti band described by Dice[3] and Blair[2], but the expression of the yellow coloration is somewhat inhibited and is considerably delayed in onset. A wild-type mouse, while it has a gray juvenile pelage, has always begun to molt into a yellow

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post-juvenile coat by three months of age, and usually even before two months. None of the whitesides, however, has ever developed the yellow color to the degree to which it occurs in the parent strains. Sometimes a very slight buffy tinge is all that appears. The oldest whiteside examined was 31 months of age, and many have been examined after one year.

Deafness appears always to be produced by the whiteside gene in double dose. A normal *Peromyscus* reacts to a squeak, such as ornithologists make to attract birds, by twitching its ears and often by jerking the entire body. Tests on normal sibs of whitesided mice indicate that they all hear this sound. Young mice first react on about the twelfth day, although the reaction becomes more pronounced after they are a week or two older. Tests on whitesides indicate that they hear the squeak from about the twelfth day until the eighteenth day. The oldest whiteside that reacted to a squeak was 24 days old. The squeak used is far from being a highly standardized sound, but nevertheless it is certain that the young whitesides have some hearing and become deaf by three or more weeks of age.

Some of the whitesided mice were observed to exhibit a type of whirling behavior similar to those previously described in *Peromyscus*[4, 7, 1]. The whirling is induced by disturbance or by strange surroundings, and may also be exhibited spontaneously. Detailed studies of this behavior were not made on the earlier mice, but with one exception no normal sibs of whitesided mice have been observed to whirl. Neither have all whitesides been observed to whirl, and both the frequency and grade of whirling seemed to increase with advancing age. Accordingly, more careful studies were commenced on the progeny of a backcross generation. Of 51 whitesides from this generation which were alive at the age of 150 days, 19 had been seen to whirl. In addition, two of seven mice from the group which died between marking age and 150 days were also observed to be whirlers. Nine of the 19 mice were, at best, low-grade whirlers, four were medium grade and six were high grade. The two which died before attaining 150 days were respectively low grade and high grade. One normal mouse in this generation was observed to whirl at 147 days and 150 days. He had appeared normal at all previous checks, and on subsequent ones at 196, 197, and 200 days. Studies on the whirling behavior are being continued, and additional information will be reported when more is known about this phenomenon.

Genetics

The circumstances surrounding the appearance of the mutant indicate that it is probably due to a single recessive gene. The presence of two abnormal mice in a sibship of eight, the consanguinity of the parents, and the absence of abnormal mice among the relatives point to recessive genes. The ratio, of course, is in exact accord with that for one gene.

Initial attempts to breed the original whitesides were not successful. They were mated to various sibs and relatives, and additional matings were made among close relatives. Only one whiteside, which died without leaving offspring, was produced by these matings. Later the two original whitesides were mated to unrelated *Peromyscus* of the *bairdi* stock and a number of wild-type F_1 mice resulted. Whitesides were recovered from the subsequent F_2 generation. Part of the whitesided mice were mated *inter se* and the remainder outcrossed again to the *bairdi* stock. Only one of the matings of whiteside $\times$ whiteside produced young which lived long enough to be classified. These four young were all whitesides. Whitesided mice recovered from the second F_2 generation were crossed to *bairdi* for the third time and the resulting heterozygotes were backcrossed to the whitesides for additional studies on segregation and other features. The breeding results are presented in Table I.

The ratio of normals to whitesides observed at marking time for the first F_2 generation is significantly different from that expected if the condition is due to a single recessive gene. The classifications had been made by temporary assistants, and some of the last litters had been discarded instead of being saved. This raised the possibility that the totals may be incorrect, either because of misclassification or of failure to record the whitesides in litters which were discarded. In the routine laboratory operations neither a classification nor a count of the offspring is attempted until the young are removed from their parents and marked. This takes place when the mice are from 25 to 31 days of age.

Accordingly, a more careful check was kept

by the writer on the second F_2 generation, including checks made at intervals prior to marking. The same data were collected on the backcross generation by Mrs. Eggleton. In addition, the latter mice were checked at intervals up to five months to determine whether additional mortality would change the ratios. These data are also given in the table. In constructing the table, all litters in which mortality had been observed before the litter had been classified have been excluded. The character can be recognized by about the third day of life, as the skin which will bear hairs with colored tips becomes very dark, whereas the areas of skin which will bear the white hairs remain pink. It is not uncommon for some mice in a litter to die within one or two days, before phenotypes can be determined. In the second F_2 generation three litters containing 13 mice were thus eliminated. Of these mice, seven were not classified, four were normal and two were whiteside. In the backcross, six litters containing 17 mice were omitted. Eleven of these animals were not classified, three were normals and three were whitesides. In addition, one other litter of two whitesides was omitted. The mother and one young died between 14 and 18 days after birth. While the remaining whiteside lived to between 40 and 43 days of age, it is likely that forced weaning at an early age had a detrimental effect. Because of the possibility that some mortality may have occurred between birth and the first observation of the litter, the totals are given in the table as "birth" ratios.

If the mice are classified into the categories normal vs. whiteside and lived vs. died for the period from birth to marking, then a contingency chi-square will indicate whether differential mortality is important. For the second F_2 generation, the adjusted χ^2 is 6.486, with P between .02 and .01; for the backcross the adjusted χ^2 is 4.278, with P between .05 and .02. It thus appears that differential mortality is acting during the period from birth to about four weeks of age. Examination of the ages at death of those mice which died before marking showed that mortality was rather evenly distributed over this period. For the backcross, the same test can be made for the interval from marking age to five months. It will be noted from the table that one normal mouse is known to have escaped, and that one whiteside was missing from its cage. It is not known whether the latter escaped or died. Even assuming that the escaped normal mouse had lived (i.e., assuming 75 normals at 150 days) and also that the missing whiteside actually died, χ^2 is only .866 (P between .50 and .30). This indicates that we cannot show any differential mortality between the ages of about one month and five months.

Finally, comparing the two F_2 segregations by the same method, we find the χ^2 is 1.989 (P between .20 and .10). Hence these two cannot be shown to differ, and we therefore have no real ground to suspect the data from the first F_2 generation.

Thus whiteside is due to a single recessive gene which exhibits essentially normal segregation at birth. Differential mortality significantly reduces the proportion of whitesides during the first month of life, but no effect was demonstrated during the period from one month to five months.

TABLE I. Segregation data for whiteside

Mating type	Normal	Whiteside	χ^2	P
wh/wh × *Wh/Wh*	191	0	—	—
wh/wh × *wh/wh*	0	4	—	—
Wh/wh× *Wh/wh*				
First Outcross				
marking age	152	29	7.781	<.01
Second Outcross				
"birth"	118	40	.008	.95-.90
marking age	112	32	.593	.50-.30
wh/wh × *Wh/wh*				
"birth"	88	73	1.398	.30-.20
marking age	81	58	1.806	.10-.05
40 days	79*	58	3.219	.10-.05
60 days	76	56	3.010	.10-.05
90 days	76	54	3.723	.10-.05
150 days	74	51†	4.232	.05-.02

* One mouse escaped during interval.
† One mouse missing from cage during interval, may not have died.

Discussion

While all gene mutations may show multiple effects if carefully studied, the present one is of interest in that it exhibits several obvious effects. Association of whirling and deafness is not surprising. The majority of whirling traits in the laboratory mouse are associated with deafness[5], although in *Peromyscus* several are not[4,7]. Association of whirling and pelage changes are known in the varitint-waddler[5] but in *Peromyscus* such combinations are not known.

It should be noted in passing that whitesided mice bear a considerable superficial resemblance to the naturally-occurring beach mouse, *P. polionotus leucocephalus*. Points of similarity are the all-white hairs on the ventrum and the sides, a considerable amount of white on the head, ears and tail tip, and finally in the general grayishness of both. Deafness and whirling behavior, however, are not characteristic of *leucocephalus*. Sumner[6] has shown that *leucocephalus* differs in pelage from the type subspecies *polionotus* by a number of genes.

Summary

Whiteside, a new recessive mutation in *Peromyscus maniculatus*, is described. The mutant phenotype has ventral hairs which are completely devoid of pigment, and the area covered by these hairs is extended irregularly dorsad for about one-half centimeter on each side. The top of the head shows some white, and pigment in the ears is reduced or lacking. The distal portion of the tail is usually white on top. The gene in double dose also delays develop-

ment of the characteristic yellow agouti band pigmentation which normally follows the post-juvenile molt, and prevents it from ever reaching normal expression. Whitesided mice are born with the ability to hear, but lose their hearing at about three weeks of age. Whirling behavior is present in some whitesided mice, appearing with advancing age, and is rare or absent in phenotypically normal sibs of whitesides. Whether this is a pleiotropic effect of the gene, or due to linkage with another gene is not yet known.

Segregation is reasonably good at birth. Considerable differential mortality occurs during the first month of postnatal-life, so that the whiteside class is numerically deficient at this time. There is no demonstrable mortality from one to five months of age. The similarity in appearance to the naturally-occurring *P. polionotus leucocephalus* is noted.

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CHEMOGENESIS?

LIVING matter—or something close to it—has been partially synthesized in the laboratory. Biochemists at the University of California in Berkeley and at Washington University in St. Louis have separated a virus into its totally inactive parts and have reconstituted it to a state of partial infective activity. The experiment has profound connotations, scientifically and philosophically, but a great distance must yet be travelled before a synthesis of "life" can be achieved.

The familiar tobacco mosaic virus, which produces a blight on tobacco leaves, was the experimental material for this work. It is a rod-shaped particle some 3,000 Angstrom units long (an Angstrom unit is one ten-millionth of a millimeter), and 140 Angstroms thick, with a hexagonal cross-section—as revealed by an electron microscope. The gross external structure of the virus is made up of protein; nucleic acid, so called because it is similar to an essential ingredient of the nuclei of living cells, runs through the rods in strands.

In the present experiments, the rods of mosaic virus were split into inert fragments, and the virus nucleic acid was separated out, apparently undamaged, by chemical means. By themselves, neither the protein nor the nucleic acid were infective; together, in a reconstituted virus, they produced the blight. But was the reconstituted product a true virus? Electron microscope photographs indicated, at least, that in the reconstructed agent the shape and arrangement of protein around the nucleic acid were like the original.

Are Viruses Alive?

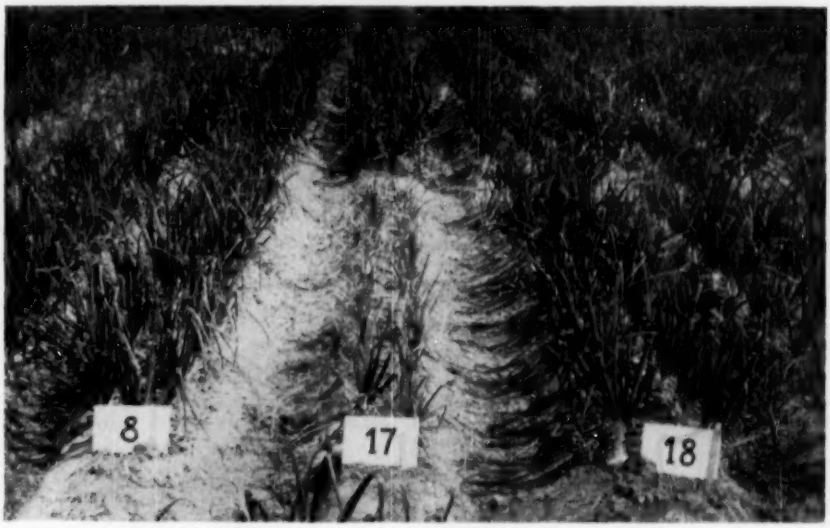
There are two schools of thought about viruses. One hold that they are not living matter, because they can be crystallized, and crystals are not normally included in our concepts of life. Moreover, they show none of the characteristics of life, such as metabolism (essentially, the burning of fuel) or reproductive activity, unless they are actually inside a living cell. But, when inside a cell, viruses can reproduce, and they can "mutate," or change their pattern of heredity; both these phenomena are considered essential properties of living matter. Viruses appear to occupy a no man's land between the large molecules of organic matter that—despite their complexity—are demonstrably not "alive," and those structures—large or small—that are capable of independent self-reproduction.

It is no surprise, in the light of biochemical research, that the building blocks of living matter should be considered as chemical entities—albeit complex. But we must now consider an ability to manipulate them—and possibly to synthesize them in the laboratory.

(Continued on page 40)

INHERITANCE OF RESISTANCE TO PINK ROOT IN THE ONION

HENRY A. JONES AND BRUCE A. PERRY*



ONIONS RESISTANT AND SUSCEPTIBLE TO PINK ROOT

Figure 12

Resistant and susceptible types of onions growing in soil infested with the pink root organism at Laredo, Texas. No. 8, resistant selection; No. 17, susceptible; and No. 18, resistant F_2. Photograph was taken March 22, 1954, and the onions were harvested on April 6, 1954.

PINK root in the onion, caused by *Pyrenochaeta terrestris* (Hansen) Gorenz, J. C. Walker, & Larson, is present in many of the important onion districts of the United States. In southern Texas the destructiveness of the disease was described by Taubenhaus and Mally[1] in 1921. The only known method for control is to develop disease-resistant varieties. Certain varieties have been developed, and selections that are highly resistant to the pink root organism have been made, but for maximum progress in a breeding program it is important to know how resistance is inherited.

Procedure

Excel, Yellow Bermuda (pink-root-resistant), Eclipse, and L 365, all Bermuda-type onions highly resistant to the pink root organism, were crossed with either Texas Early Grano 951 or San Joaquin, both very susceptible. All crosses and selfs were made in the greenhouse at the Plant Industry Station, Beltsville, Maryland. Seed of the backcross populations was planted in the greenhouse at Beltsville on September 6, 1954. The plants were pulled on November 26, taken to Laredo, Texas, and planted shortly after arrival in soil infested with the pink root organism. All other seed was planted in late September at Crystal City, Texas, in soil free from the pink root organism and transplanted in late November in soil infested with the pink root organism at Laredo, Texas. The test was on the Norman Clark farm

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(near Laredo) on soil that had been used for pink-root trials for four years and was uniformly infested with the fungus. The plants were grown on raised beds, two rows to the bed, and furrow irrigation was used.

Results

Harvesting was done on March 23 and 24, 1955. At this time the tops were still green but beginning to fall, indicating maturity. When the F_3 lots were pulled, the roots of some of the plants were white and offered considerable resistance on being lifted. Other plants were easy to pull and had roots with slight infection to almost complete destruction. It was not difficult to classify the plants into resistant and susceptible types. All the backcross plants from the Texas Early Grano 951 parent were badly infected, the plants were easy to pull, and only small bulbs were produced.

In the F_3 populations the resistant plants usually produced bulbs two inches or more in diameter so that it was possible to select for bulb type. As a rule, most of the susceptible plants formed no bulbs or small ones.

Table I gives the results of the tests for the 1954-55 season. The chi-square test shows a satisfactory 3:1 ratio of susceptibles to resistants in the F_3. However, the (L 303 × L 365) T.E.G. 951 F_3 population had a rather poor fit. No resistant plants appeared in the backcross population or in the F_1 hybrid.

Texas Early Grano 951, a susceptible parent, had practically all roots destroyed, but Eclipse, one of the resistant parents, had an extensive root system with almost no indication of infection. Figure 12 shows clearly the good growth of the resistant types and the uniformly poor growth of one of the susceptibles when grown on soil heavily infested with the pink root organism.

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TABLE I. Performance of parents and various progenies on soil infested with the pink root organism, Laredo, Texas, 1954-55

Pedigree	Number of populations	Generation	Resistant plants	Susceptible plants	χ^2	P
			No.	No.		
(L 303 × L 365) Texas Early Grano 951	17	F_3	303	1,068	6.1470	0.01
Texas Early Grano 951 × Yellow Bermuda (pink-root-resistant)	18	F_3	307	987	1.1220	.20
Excel (986) × Texas Early Grano 951	12	F_3	252	780	.1860	.50
Excel (986) × San Joaquin	1	F_3	54	167	.0377	.95
Texas Early Grano 951 [(L 303 × L 365) Texas Early Grano 951]	6	Backcross	0	119	.....	...
Excel (986) × Texas Early Grano 951	1	F_1	0	160	.....	...
Texas Early Grano 951	1	...	0	160	.....	...
Eclipse	1	...	160	0	.....	...

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ACTIONS OF RADIATIONS ON LIVING CELLS

THE first edition of this book* published in 1946 has long been the standard for this subject. It was written by D. E. Lea, a physicist, and is quite comprehensive in its scope, as is seen by a glance at the table of contents: Chapter 1, Physical properties and dosimetry of different radiations; Chapter 2, Chemical effects of ionizing radiations and possible mechanisms of biological action; Chapter 3, The target theory; Chapter 4, The inactivation of viruses by radiation; Chapter 5, Genetical effects of radiation; Chapter 6, The production of chromosome structural changes by radiation; Chapter 7, The mechanism of induction of chromosome structural changes; Chapter 8, Delayed division; Chapter 9, Lethal effects. In the 1946 edition there was one appendix on supplementary calculations.

The topic "Actions of Radiations on Living Cells" is a vital one and one that changes rather rapidly. For this reason, the 1946 edition is out of date in some respects. For example, nothing is included regarding the effect of oxygen in altering the effectiveness of ionizing radiations. Also, there is nothing included regarding the protective substances

**Actions of Radiations on Living Cells.* (Second Edition) D. E. Lea. The University Press, Cambridge, England, 1955. 374 p. $6.00.

against ionizing radiations. At the time the 1946 edition was published little was known regarding the sensitivity of the different stages in the development of the germ cells to ionizing radiations. Considerable evidence has accumulated in plants and animals showing that there are tremendous differences in susceptibility to radiations according to the different stages in the meiotic cycle. In both corn and *Drosophila* the sensitivity increases as the germ cells near maturity, and then there is a sharp decrease in the mature germ cells.

Since the first edition was published in 1946, better dosimetry for neutron irradiation has been developed, and neutrons have been shown to be more effective than X-radiation biologically. However, the 1946 edition stated that the X-rays were more effective.

For these and other reasons it seems that a thorough revision of Lea's book was in order. However, the 1954 edition is not really a revised edition but a reprint of the old one. Page for page it is identical with the older edition. The only difference in the new one is a second appendix containing limited data of a few of the more recent findings. However, nothing really recent is in this appendix, which was prepared largely by the late Dr. D. E. Lea for revision of the first edition.

The remainder of the revisions were made by L. H. Gray, one fully competent to have done a thorough revision of the original edition. There is also a short addition to the bibliography but the author index and the subject matter index are identical with those for the earlier edition. For anyone possessing the earlier edition of Lea there is not much point in having this one. For anyone not having the first edition it would probably be well to have this book, bearing in mind that it is considerably out of date.

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BREEDING BEEF CATTLE FOR UNFAVORABLE ENVIRONMENTS

AMERICA is a new country, yet one cattle breeding establishment celebrated the completion of 100 years of continuous operation by members of the same family, in 1953. This in itself is noteworthy, but when the fact is considered that a new breed of beef cattle has been developed here on this tremendously large property, the achievement is unique in the annals of this country. The personnel of the King Ranch have worked in close cooperation with Experiment Stations, Colleges and the U.S. Department of Agriculture on many technical aspects of ranch management and beef breeding and production. Recognition of this is implied in the expression by Robert J. Kleberg, Jr. as follows: "We wanted to mark the centenary of the ranch as a livestock operation, not by a big parade, or pageant, or a 'blow-out', but by something that would be representative of your efforts and our efforts. And we wanted to give recognition to the industry and to the animal sciences which have contributed so much to the progress of this ranch." Pursuant to this idea the King Ranch invited something over 100 men and their wives to be their guests Oct. 17-20, 1953 during which time a symposium on "Breeding Beef Cattle for Unfavorable Environments" was presented.* Speakers representing nutrition, climatology, soil fertility, animal health, meats, plant breeding and animal breeding contributed from nine states, District of Columbia, Europe, South Africa and South America. Average attendance was 150 persons, including members of faculties of sixteen North American colleges or universities, Cambridge University (England), the University of Sao Paulo (Brazil), and the University of Adelaide (Australia). Agricultural technicians and cattle breeders were present from Argentina, Brazil, Colombia, Cuba, Venezuela, and the Union of South Africa, as well as from the cattle-raising areas of the United States. A few ranchers, newsmen, and friends of the King Ranch were the other participants. Four field trips added color and general interest to the occasion, but special interest centered on the Santa Gertrudis cattle.

The emphasis in the symposium was put on the fact that most cattle originating in northern climates degenerate in the warmer regions of the earth. Much of the discussion concerned the causes of this, whether due to nutrition, soil fertility, poor heat tolerance, or undermined health due to inroads of disease. Discussions of the theoretical aspects of applied genetics was followed by discussions of attempts to improve cattle in several parts of the world, including a review of the procedures used in developing the Santa Gertrudis breed at King Ranch.

The formal papers have been brought to-

**Breeding Beef Cattle for Unfavorable Environments.* Albert O. Rhoad (Editor). A Symposium Presented at the King Ranch Centennial Conference. 248 pp. Univ. of Texas Press, Austin. 1955. $4.75.

gether in a volume published by the University of Texas Press. It would be impossible to give adequate review of these papers in the space at our disposal. The crystalization of the ideas and viewpoints in the various fields represented in these papers make this volume a valuable contribution. It will be of interest to the general reader as well as to the specialists working on cattle breeding problems. The King Ranch and the University of Texas Press are to be commended for making this excellent publication available.

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Counseling in Medical Genetics

IN the past many physicians have considered genetics to be a hopeless aspect of medical practice to be neglected or avoided. The genes of their patients could not be changed, they told themselves, so why bother with the unpleasant subject and its implications. The trend has changed in recent years. A knowledge of the principles and applications of genetics has now been found to aid significantly in diagnosis, prognosis, management and prevention of disagreeable symptoms associated with several human ailments. Many medical schools are now finding places in the crowded curriculum for courses in medical genetics.

The counseling aspect is perhaps the most important to the physician. Dr. Sheldon C. Reed* has done much to aid the physician in this area by bringing together the pertinent data and supplying interpretations covering the traits about which the general practitioner is most frequently consulted. Excellent training in fundamental genetics and wide experience as Director of the Dight Institute for Human Genetics at the University of Minnesota have fitted Dr. Reed admirably for this task. His sympathetic understanding of people and appreciation for their problems have made him an excellent example for the physician to follow in counseling.

The purpose of the book is to help the physician answer his patient's questions concerning heredity. Most of the book is devoted to a consideration of the 20 most common causes for counseling at the Dight Institute. These traits are discussed concisely but adequately for most purposes. Each of the 20 traits included appears with a frequency of better than one in 1,000 births in the general population. Twin data are used wherever available to establish the heritability of the trait under consideration. Risk figures are also presented in understandable terms for various situations. The type and validity of the data upon which the risks are based are critically evaluated.

For example, the discussion on rheumatic fever begins with a listing of the reasons for conflicting opinions regarding the genetic factors involved. A general statement follows concerning the importance of the disease in different parts of the world and its relationship with heart disease. A summary of 246 twin pairs reported by a dozen different groups of investigators shows that among the 113 pairs of identical twins, 32 (28 percent) both had rheumatic fever and 81 pairs had only one member affected. Among the 133 pairs of fraternal twins, 10 (eight percent) had both affected while 123 had only one member affected. The sex-association is next pointed out from statistical data showing girls to be more susceptible than boys. The statistical risk is evaluated at 9 percent if neither parent is affected and 19 percent if one parent is affected. The risk for each child of an affected person for eventual involvement is 12 percent.

A well chosen illustrative example from an actual counseling experience at the Dight Institute concludes the chapter on rheumatic fever. Similar illustrative examples follow the other chapters and represent a most interesting and valuable part of the book. In addition to the 20 subjects treated in some detail a large number of rare traits are listed with pertinent notes in the appendix. Brief but pointed discussions of the basic principles of inheritance and the philosophy of counseling are also included. The purpose set forth by the author has been accomplished as well as possible for a work of small size.

The book is highly recommended for physicians and others who are called upon for counsel in human genetics. It is readable, concise and well organized. The indexing is excellent making it suitable for quick reference. The literature cited section includes references to the numerous original investigations from which the facts used in the book were obtained. Physicians who are not technically trained and widely experienced in human genetics counseling will appreciate having this book within arms reach on their desks.

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**Counseling in Medical Genetics.* Sheldon C. Reed. W. B. Saunders Company, Philadelphia and London. 1955. viii + 268 pp. $4.00.

SILKY PLUMAGE IN THE RING NECK DOVE

WILMER J. MILLER*

A BLOND, SILKY MALE

Figure 13

J1.1 is the "original" silky, a blond male dove (*L*/+). The feathers are plumose and regular in this "moderate" grade of silkiness. *Photograph by Hugh Wilmar.*

A SHIPMENT of ring neck doves, *Streptopelia risoria*, was received from a California dealer in November, 1951. It was noted that one male dove (Figure 13) possessed abnormal plumage greatly resembling "silky" of the domestic pigeon. Cole and Hollander[2] reported that in the domestic pigeon, *Columba livia*, "silky" or "lace" was an autosomal dominant (*L*) of two grades, moderate and extreme, corresponding to the heterozygous and homozygous state, respectively.

The male dove with "silky" plumage was immediately placed in mating and except for a period of illness, it produced offspring regularly, until it died in the Genetics Barn fire of July 1955. From

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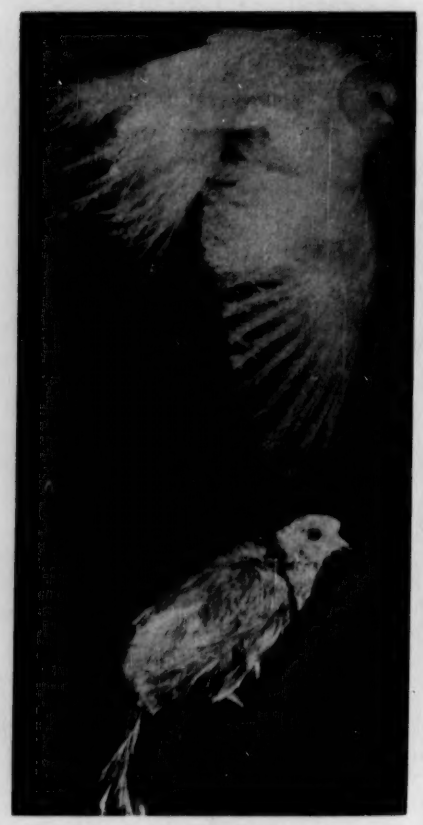


MODERATE AND EXTREME SILKIES

Figure 14

A—Shows a "moderate" silky bird (*L*/+), the white great-grandson of J1.1 shown in Figure 13. *B*—"Extreme" silky (*L*/*L*), the blond granddaughter of J1.1.

matings of its offspring and their progeny of silky by silky, offspring of two grades of silkiness were evident—moderate and extreme. The feathers of moderate silkies (Figures 13 and 14*A*) are plumose and regular as in the "original" silky dove (J1.1); whereas the feathers of the extreme silkies (Figure 14*B*) appear tattered and irregular.

Breeding and Experimental Data

In successive matings with six normal ring neck females, the silky ring neck dove (J1.1) has produced a total of 24 silky and 14 normal offspring, indicating that the trait is dominant and that the male was heterozygous. All immediate offspring closely resembled the silky parent, or else were normal. Some silky progeny in turn were mated with normals and some with one another. As indicated in Table I, 71 silky and 60 normal offspring have been obtained from matings of moderate silky to normal birds. From matings of silky with silky, 12 offspring were "extreme" silky (Figure 14*B*); 27 were "moderate" silky similar to the "original" mutant; and 19 were normal. The numbers of offspring in the different classes do not deviate significantly from the 1:1 and 1:2:1 ratios characteristic of families segregating for a single dominant gene.

Since the female is the heterogametic sex in pigeons and doves, the pertinent matings to determine whether or not the gene for silky is sex-linked, are those in which the female parent is silky and the male normal. All male offspring should be silky and all female offspring should be normal. Six such matings had a total of 46 classified offspring. Of 25 females, 16 were silky and nine normal; of 21 males, 14 were silky and seven normal. Therefore, there is no evidence of sex-linkage. (One family has biased the ratio of silkies to normals.)

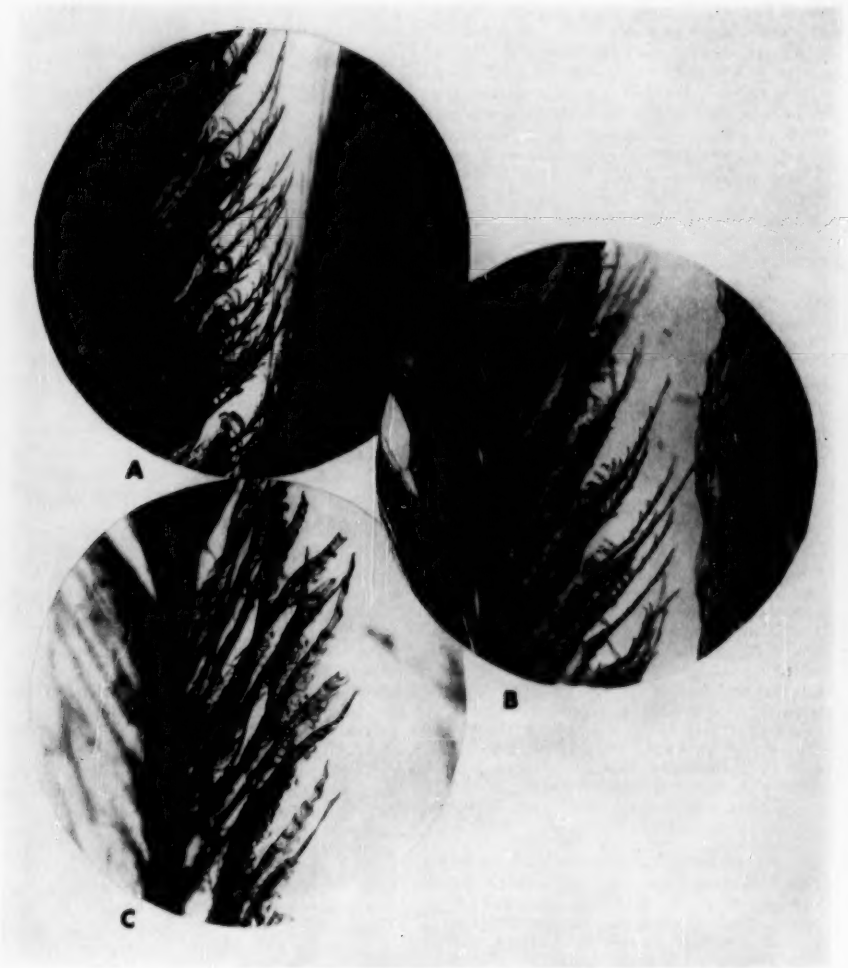
The effect of the silky factor in pigeons is apparently a reduction in strength and elasticity of the barbules of the feather, and a tendency of the barbs to twist. The down of nestlings also tends to curl[2]. Steele[3] noted a similarity in the general appearance of silkiness in the chicken and in the pigeon, although in chickens, silky is recessive instead of dominant as in the pigeon. Moreover, he stated that silky pigeons have hooks on the barbules, whereas the feathers of the silky chicken lack hooks.

Microscopic examination shows that the feathers of moderate, and extreme silky doves closely resemble those pictured by Cole and Hollander[2] for silky pigeons. As in silky pigeons, the silky dove possesses hooks on the barbules but they seem abnormal in form under a higher power (100×) than pictured by Cole and Hollander. (See Figure 15*B* and *C*).

The hamuli or hooks on the barbules are thickened and somewhat crowded compared to the normal feather. The barbule appears to

TABLE I. Breeding data of silky ring neck doves

Number of Matings	Mating Type of Parents	Number of offspring in class: Extreme Silky (*L*/*L*)	Moderate Silky (*L*/+)	Normal (+/+)	χ^2	P
16	moderate silky × normal	0	71	60	.92	.31
10	mod. silky × mod. silky	12	27	19	1.96	.38
2	extreme silky × normal	0	7	0		



NORMAL, MODERATE AND EXTREME SILKY PLUMAGE

Figure 15

Magnified views of normal (*A*), moderately silky (*B*), and extremely silky (*C*), demonstrating the homozygous normal, the heterozygous (*L*/+), and the homozygous dominant (*L*/*L*) silky respectively. (100 ×). Note the twisting and terminal breaks of the barbules of the homozygous silky. All views are focused on the distal side of barbs of secondary feathers. *Microphotographs by Dr. Katherine Beamish.*

have structural "lines" more or less perpendicular to the length of the barbule and at which the barbule may break (Figure 15*C*). Such "fracture lines" are especially noticeable in the extreme silky feather in which the barbules themselves seem shortened, perhaps because the terminal portion has broken off.

The author was not successful in predicting moderate silkiness from examination of the down of nestlings. However, extreme silkies had practically no down at all as squabs. The vanes of the feathers, especially of the rectrices and remiges, of the extreme silky tend to break off, leaving the shaft projecting. Moderate

silkies are unable to fly more than a few feet on level surfaces and are easily recovered by hand. Extreme silky males have been unsuccessful in attempts to copulate with silky or normal females, but extreme silky females are fertile. An undove-like tendency to "back-up" has been noticed occasionally in the extreme silkies. As yet, the author has not been able to find the cause of this peculiar behavior. Even in new plumage the extreme silky cannot fly.

Silky may be combined with other mutants such as the three sex-linked alleles, "white" (Figure 14*A*), "blond" (with which the silky trait first occurred) and the wild-type "dark."[1] The heterozygous silky dove is quite attractive, and since it is smaller and easier to keep than the domestic pigeon, it may become a popular garden or house pet of fanciers. Silky doves may be kept as a producing pair of cage birds inside the house or laboratory in one of the larger canary or parakeet cages.

Summary

An autosomal dominant, "silky" plumage, is reported for the ring neck dove, *Streptopelia risoria*. Two grades, moderate and extreme, corresponded respectively to the heterozygote (*L*/+) and the homozygote (*L*/*L*). The similarity to silky in pigeons is noted.

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CHEMOGENESIS?

(*Continued from page 32*)

The protein components of broken viruses, measured during the reconstitution experiments, have a molecular weight of 100,000, and seem to be composed of sub-units of molecular weight 20,000. Biochemists have already achieved controlled synthesis of proteins up to a molecular weight of 800, and it might become possible through further developments in technique to synthesize the sub-units and fuse them into the larger protein units used to reconstitute the virus.

The long-range possibilities are far-reaching. We may be able, for example, to produce "tailor-made" viruses, by breaking them up and reconstituting them in a form that would give immunity to the disease induced by the original. Antigens, developed from virus fragments, might be used to stimulate the production of antibodies, which fight disease. Or it may become possible to synthesize viruses in the laboratory out of inert materials; so far, however, we have not learned to analyze completely, let alone synthesize, the nucleic acids.

Synthesis of a virus, if achieved, would be only a faltering first step toward the synthesis of life, as we are accustomed to think of it. Even the past 50 years of analytical biochemical research have given at best only a gross idea of the complexity of a living cell. We know the structure of only a few of the hundreds of different proteins in a cell; how these combine with nucleic acids, proteins, and other components to form the cell structure is at present pure conjecture. Viruses, the simplest form of life,—if that—offer at best a preliminary training ground in biochemical technique.

Reconstitution of the tobacco mosaic virus does, however, represent a new approach to our understanding of heredity. In a sense, a virus is a kind of genetic agent; in its composition, replication, mutation, and determinative effect on cell development, a virus resembles a chromosome, the carrier of hereditary characteristics. The synthesis of one self-duplicating system introduces the possibility of studying and manipulating others, such as chromosomes from higher organisms.

Whatever the direction of future work, the reconstitution experiments represent a long step forward in virus research, and a significant milestone toward our attempts to understand the basic phenomena of life.

—*Industrial Bulletin* of Arthur D. Little, Inc.
January, 1956

INTRA-SPECIES RELATIONSHIPS AND LYSENKO'S CONCEPT OF A SPECIES

CONSTANTINE SOROKIN AND W. GORDON WHALEY*

A DISTINCTION between intra- and inter-specific relationships in the definition of the concept of a species was suggested by T. D. Lysenko in 1948 and elaborated by him later, in 1950.

> *"A species is a specific, qualitatively distinct state of living form of substance. Essential features of species of plants, animals, and microorganisms are certain intra-species relationships among individuals.* (Lysenko's italics) These intra-species relationships are qualitatively different from those between individuals of different species. *Therefore, a qualitative difference between the intra- and inter-species relationships is one of the most important criteria for distinguishing species forms from varieties."* (Lysenko's italics) (p. 19)[2]

To make the difference between intra- and inter-species relationships clear, Lysenko explains:

> "There is not, and cannot be, a class society in any plant or animal species. Therefore, there is not, and cannot be, here class struggle, though it might be called, in biology, intra-species competition." (p. 552)[1]
>
> ". . . in nature the life of any individual is subjected to the interests of its species." (p. 604)[1]
>
> ". . . all intra-species relationships among individuals, just as those of organs of an organism, are directed toward the securing of the existence and thriving of a species and this means, toward the increasing of the number of the individuals of a species." (p. 662)[1]

Among the evidence brought forth by Lysenko to prove the absence of intra-species competition a prominent place is occupied by consideration of experiments conducted with kok-saghyz (*Taraxacum kok-saghyz* Rodin) in Moscow in 1945 (p. 523-57)[1]. In these experiments plants were grown in hills spaced 40 × 60 cm. apart. In the first experiment plantings were made with 100-200 seeds in each hill; in the second experiment, with small pieces (0.2-0.3 gm. each) of roots, 8-15 pieces to a hill.

By the time of harvesting there were different numbers of plants in different hills and, since the distance from one hill to another was considered by the author large enough to exclude the influence of plants of one hill upon those of another, plants in a particular hill grew, in Lysenko's opinion, only under the group influence of the other plants of the same hill.

This influence of one plant upon another was in particular revealed in the different and much smaller number of plants in each hill at the time of harvest as compared with the number of seeds sown, and in the different development (weight) of plants in a certain hill. What Lysenko tries to prove is that all these signs of mutual influence of plants of one hill are expressions of factors other than the competition among the plants of a certain hill.

The results of Lysenko's second experiment are presented in Table I. The data are arranged in horizontal rows according to diminishing weights of the plants. If these data are followed horizontally then, Lysenko concedes, they give an impression of the existence of the competition among plants of the same hill.

> "On the whole, these data look as though they entirely prove the existence of the intra-species competition,

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the oppression of each weaker plant by a stronger one. It is possible to think that this oppression continues to the complete elimination, eradication, of some plants by the others. Watching kok-saghyz plants from seedlings until maturity, it is easy to see a continuous decrease in the number of individuals, the self-thinning.

"But the conclusion on the existence of the intra-species competition . . . is entirely wrong." (p. 543-544)[1]

To prove the absence of the intra-species competition Lysenko analyzes these data in vertical rows. The average weight of the one root in hills in which, by the time of harvesting, there was only one plant, was 65.8 gm.; in the hills with the larger number of plants the weight of the heaviest plant, it is true, was less, but the decrease in the weight of the heaviest plant went only to a certain point. In the hills with eight plants the average weight of the heaviest plant (34.4 gm.) was essentially equal to the average weight of the heaviest plant in the hills with 13 plants (34.2 gm.).

The second heaviest root in the hills with only two plants weighed 28.4 gm.; the comparable root in the group of hills with 13 plants in a hill was nearly as heavy—23.7 gm.

Beginning with the third heaviest root in a hill, the roots were generally heavier in the hills with the larger number of plants. For instance, in the group with four plants in a hill the average weight of the fourth heaviest roots was 11.2 gm., and in the group with 10 plants in a hill it was 16.1 gm. For the fifth heaviest roots the average weight in the group with five plants was 9.2 gm., that in the hills with 10 plants—13.1 gm.

The fourth heaviest roots in the hills with four plants grew in the neighborhood of roots totaling 87.6 gm., while roots of the fourth heaviest plants in the hills with 13 plants sustained the oppressing influence of 153.5 gm. of roots of neighboring plants. Despite this fact the weight of roots in the first group was 11.2 gm., and that in the second group, 18.0 gm.

"Where is in this case the oppression, and, moreover, competition? There is nothing of that kind." (p. 545)[1]

Actually, Lysenko's data not only furnish a good example of the existence of the competition among plants within a hill (intra-species competition), they also give suggestions as to the nature of the relationships among the competing individuals.

Even the mere fact, mentioned by Lysenko himself, of the decrease in the number of plants in a hill during development from seedlings to maturity suggests that the most plausible explanation of the elimination of some plants (together with the effects of diseases and unfavorable growing conditions) is a deleterious influence exerted by the stronger plants on the weaker ones, this influence finally leading to the death of some of the latter.

Another proof of the unfavorable effects exerted by one plant upon the other, and of the increase of these deleterious effects with an increase in the number of the plants in a hill, is the average weight of the roots of one plant in a hill. Using Lysenko's data, average root weights can be calculated as shown in Table II.

TABLE I. Planting of kok-saghyz with pieces of roots

Number of plants in a hill	Number of hills	Average total weight of roots in a hill	Average weights												
			Heaviest root in a hill	2nd heaviest root in a hill	3rd heaviest root in a hill	4th heaviest root in a hill	5th heaviest root in a hill	6th heaviest root in a hill	7th heaviest root in a hill	8th heaviest root in a hill	9th heaviest root in a hill	10th heaviest root in a hill	11th heaviest root in a hill	12th heaviest root in a hill	13th heaviest root in a hill
1	78	65.8	65.8	—	—	—	—	—	—	—	—	—	—	—	—
2	190	79.8	51.4	28.4	—	—	—	—	—	—	—	—	—	—	—
3	297	92.4	48.4	27.0	17.0	—	—	—	—	—	—	—	—	—	—
4	401	98.8	43.7	26.1	17.7	11.2	—	—	—	—	—	—	—	—	—
5	402	114.2	42.6	27.7	20.0	14.6	9.2	—	—	—	—	—	—	—	—
6	316	117.7	38.3	25.1	19.1	14.9	11.8	8.2	—	—	—	—	—	—	—
7	273	128.8	38.5	25.7	19.8	15.5	12.2	9.9	6.8	—	—	—	—	—	—
8	115	110.0	34.4	24.7	19.7	14.9	12.2	10.5	8.0	5.3	—	—	—	—	—
9	74	135.7	34.6	23.6	17.4	14.9	13.2	10.4	9.0	7.1	5.1	—	—	—	—
10	24	143.2	33.9	21.4	17.4	16.1	13.1	11.1	9.6	7.6	6.9	5.6	—	—	—
11	18	121.5	29.7	21.1	15.2	11.1	9.3	9.7	7.2	6.1	5.7	5.4	3.1	—	—
12	5	143.4	35.6	19.8	17.2	13.6	12.0	10.0	7.0	7.0	6.6	5.8	5.2	3.6	—
13	4	171.5	34.2	23.7	19.2	18.0	13.2	10.7	10.5	9.5	8.0	7.5	7.5	5.0	4.2

There is clearly a pronounced decrease in the average weight of roots of the plants with the increase in the number of plants in a hill. This observation is not modified by the apparent increase in the root weight in the hills with 12 and 13 plants, in which cases only a small number of hills were available.

The weakening effect of the competition among members of one hill is also shown by the decrease in the weight of the strongest plant (assuming the heaviest roots to be those of the strongest plants) in hills with larger numbers of plants (Table I). There is, it is true, a diminution in the rate of this decrease as one proceeds from the hills with small numbers of plants to those with a larger number. But, the dependence of the development of the particular plant on the number of plants (i.e., the size of the population) in a hill should not be permitted to obscure the existence of a competition among the plants of one hill.

As the number of plants in a hill increases, the increase is largely due to the increase in the number of very weak plants. The existence of such plants adds only a little to the bulk of the competing resistance which members of the group, among them the strongest, have to oppose in their struggle for existence.

In the universal competition among organisms each has to struggle against those which are stronger, those which are equally strong, and, also, to a varying extent, against those which are weaker. If we compare, for instance, the strongest plants in the hills with eight plants with those in the hills with 12 or 13 plants we see that the average weight of the strongest plant did not decrease with the increase in the total number of plants and in the total mass (weight) of the roots in the hill. This is due to the fact that the number of plants and the mass of roots in the hills with 12 and 13 plants as compared with those with eight plants increased chiefly on account of very weak plants. But, weaklings do not constitute a serious peril to the existence or well-being of the stronger individuals. Actually, the influence of weaker plants reaches, with increasing weakness, a vanishing point. That this suggestion is true can be proved with the examples chosen by Lysenko himself.

If the average root weights of the fourth strongest plants in the group of hills with four and 10 plants in a hill and the average root weights of the fifth strongest plants in the groups of hills with five and 10 plants in a hill are compared, not with the average total root weights of all plants in the corresponding groups of hills, but separately with the average total root weights of plants which are stronger or weaker than those plants chosen for consideration, then the picture will appear quite different from that presented by Lysenko.

From Table III it is seen that the weights of the fourth and the fifth heaviest roots (column 1) seem to be favored (increased) by the increase in the mass of the other roots in the hill (column 4). An explanation to this paradoxical observation is found in the fact that in passing from hills with four and five plants to those with 10 plants the increase in the mass of all roots in the hill is accounted for by the appearance of a number of plants which are weaker (column 3) than the fourth or the fifth strongest plants. Parallel to the appearance of the weaker plants there is a decrease in the mass of the roots of the stronger plants (column 2) in a hill.

TABLE II. Average weight of roots in a hill

No. of plants in hill	No. of hills	Avg. wgt. of roots
1	78	65.8
2	190	39.9
3	297	30.8
4	401	24.7
5	402	22.8
6	316	19.6
7	273	18.4
8	115	16.3
9	74	15.1
10	24	14.3
11	18	11.2
12	5	11.8
13	4	13.2

TABLE III. The dependence of the weights of the fourth and the fifth strongest plants on the development of the stronger plants in the hill

	Avg. root wgts. of the fourth strongest plant. 1.	Avg. total root wgts. of the three stronger plants. 2.	Avg. total root wgts. of all weaker plants. 3.	Avg. total root wgts. of both stronger and weaker plants (2+3). 4.
In the hills with four plants.	11.2 gm.	87.5 gm.	0.0 gm.	87.5 gm.
In the hills with ten plants.	16.1 gm.	72.7 gm.	53.9 gm.	126.6 gm.
	Avg. root wgts. of the fifth strongest plant. 1.	Avg. total root wgts. of the four stronger plants. 2.	Ave. total wgts. of all weaker plants. 3.	Avg. total root wgts. of both stronger and weaker plants (2+3). 4.
In the hills with five plants.	9.2 gm.	104.9 gm.	0.0 gm.	104.9 gm.
In the hills with ten plants.	13.1 gm.	88.8 gm.	40.8 gm.	129.6 gm.

Thus, for a particular plant in the hill, development depends not on the number or strength (as measured here by root weight) of all plants in the hill but mostly on the number and strength of those plants which are stronger. Most important for the well-being of an individual is its ability to resist the combined competing power of all stronger organisms and, to an extent, of those which are somewhat weaker. The existence of very weak organisms does not add much to the resistance which an individual has to exert in intra-species and inter-species competition.

To show that these conclusions extend not only to the chosen examples but are also of a more general nature, the above-demonstrated relations are presented as correlation coefficients calculated for pairs of rows computed as it is shown in the example given in Table IV. In the second column of the table are given average weights of particular plants (in the example, of the third heaviest plants) in hills with different numbers of plants in a hill (column 1). In the third column are given the average weights of all plants in the hill which are stronger (heavier) than the plants whose average weights appear in the second column. Only groups of hills with not more than 10 plants in a hill were taken for the calculations since the number of hills counted in groups with larger number of plants in a hill was much smaller, and, hence, the average data are not so reliable as those for groups with 10 or fewer plants per hill.

Correlation coefficients calculated from Lysenko's data computed as shown in Table IV, are given in Table V.

High negative correlation coefficients between the root weights of a particular plant and the root weights of all plants which were heavier than it, show that in its development a particular individual is restricted by those in the same group which are stronger (heavier roots). For the seventh and eighth heaviest plants these relations assume a form of a negative function, so close are correlation coefficients to unity. As one proceeds from these comparatively weak plants to those which are stronger (the sixth, fifth, and fourth plants) one notices that the dependence of the development of these plants on those which are stronger is high but not so high as in the case of the seventh and eighth plants.

This situation is evidently due to two factors. First, as we proceed from the weaker plants in a hill to those which are stronger, we notice that the combined oppressive power of all plants which are stronger than this particular plant becomes less and less. Second, with the decrease in the oppressive power of plants which are stronger, the competing power of those plants which are weaker grows progressively.

The correlation coefficient for the third heaviest plant, which is close to zero, indicates that this plant in its development was restricted by stronger plants (of which there were only two) essentially no more than by those which were somewhat weaker. Here, there were many of them in the hill, and the fourth and fifth strongest plants, for instance, were strong enough to influence the third heaviest plant.

A positive correlation coefficient for the second heaviest plant ($r = +0.86$) does not prove that its development was favored by the development of the stronger, in this case of the first strongest plant. It simply shows that the oppressive power of only one, even the strongest plant, was not enough to make the second heaviest plant depend only on the development of the stronger plant. A high correlation coefficient between the weight of the second and that of the first heaviest plants indicates that they were in a similar position. The third, the fourth, and even some still lesser plants in the hill were vigorous enough to be strong competitors. They markedly influence both the first and the second heaviest plants.

Analysis of Lysenko's data proves that all members of a group develop under the competing influence of the other members of the group. The competing relationships between two members of the group depend not only upon their relative strength but also on the competing influence of the other members of the group and, therefore, on the size of the population. Surely, Lysenko's data present clear cut evidence for the existence of intra-species competition, the existence of which he has denied.

TABLE IV. Sample computation of the correlation coefficient between the weight of the third heaviest root and the total weight of the (two) roots which are heavier than the third heaviest root

No. of plants in a hill.	Avg. root wgt. of the third heaviest plant.	Avg. root wgt. of all plants in the hill which are heavier than that in column 2.
3	17.0	75.4
4	17.7	69.8
5	20.0	70.3
6	19.1	63.4
7	19.8	64.2
8	19.7	59.1
9	17.4	58.2
10	17.4	55.3

TABLE V. Correlation coefficients between the root weights of a particular plant and the root weights of all plants which are heavier than it

For the	2nd	strongest	plant	$r = +0.86$
" "	3rd	"	"	$r = -0.04$
" "	4th	"	"	$r = -0.56$
" "	5th	"	"	$r = -0.90$
" "	6th	"	"	$r = -0.67$
" "	7th	"	"	$r = -0.98$
" "	8th	"	"	$r = -0.99$

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